UNIVERSITY OF MARYLAND
LIBRARY
Founded 1813
SCHOOL OF MEDICINE

HENRY M. BOLLMAN LIBRARY
UNIVERSITY OF MARYLAND
BALTIMORE

Digitized by the Internet Archive
in 2016

https://archive.org/details/journalofkansasm5511kans

The Journal

OF THE KANSAS MEDICAL SOCIETY

NONTRANSFERABLE

UNIVERSITY OF MARYLAND
LIBRARY
Founded 1813
JAN 20 1954
SCHOOL OF MEDICINE

In This Issue

THE TREATMENT OF DIABETIC ACIDOSIS AND DIABETIC COMA—Edward Tolstoi, M.D., New York, New York . . . . . Page 1

MANAGEMENT OF ACUTE LARYNGOTRACHEO-BRONCHITIS—Harold L. Hickey, M.D., Denver, Colorado . . . . . Page 7

THE DIAGNOSIS OF LEAD POISONING—John L. Morgan, M.D., Emporia, Kansas . . Page 11

THE EFFECT OF ALTITUDE ON THE PATHOGENESIS OF TUBERCULOSIS—William F. Casteen, Kansas City, Kansas . . . . . Page 16

COMPLETE TABLE OF CONTENTS . PAGE IV

JANUARY, 1954 VOL. LV, NO. 1

SURITAL® SODIUM

ULTRASHORT-ACTING INTRAVENOUS ANESTHETIC

SURITAL sodium (thiamylal sodium, Parke-Davis) produces smooth anesthesia with rapid, quiet induction and prompt, pleasant recovery.

Detailed information on SURITAL sodium will be mailed you on request.





MEDICAMENTA VERA

Parke, Davis & Company

DETROIT, MICHIGAN

TABLE OF CONTENTS

JANUARY, 1954

ORIGINAL ARTICLES

The Treatment of Diabetic Acidosis and Diabetic Coma—Edward Tolstoi, M.D., New York, New York 1

Management of Acute Laryngotracheobronchitis—Harold L. Hickey, M.D., Denver, Colorado 7

The Diagnosis of Lead Poisoning—John L. Morgan, M.D., Emporia, Kansas . . . 11

The Effect of Altitude on the Pathogenesis of Tuberculosis—William F. Casteen, Kansas City, Kansas 16

EDITORIALS

Polio Vaccination Field Trials 21

VA Non-Service Connected Cases 22

The Facts of Life 23

Changes in A.M.A. Dues Policy 23

MISCELLANEOUS

President's Page 20

Adenocarcinoma of the Prostate—Tumor Conference 25

The Month in Washington 37

Radioactive Iodine in Hyperthyroidism—Senior Thesis 40

Entered as second-class matter at the post office at Fulton, Missouri, June 19, 1953, under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of October 3, 1917, authorized on July 2, 1918. Office of Publication: 1201-05 Bluff Street, Fulton, Missouri.

THE JOURNAL OF THE KANSAS MEDICAL SOCIETY

Editorial Board

ORVILLE R. CLARK, M.D., *Editor,* Topeka
JOHN W. CAVANAUGH, M.D., Topeka
DAVID E. GRAY, M.D., Topeka
RICHARD GREER, M.D., Topeka
DWIGHT LAWSON, M.D., Topeka

Associate Editors

DONALD P. TREES, M.D., Wichita
GLEN R. SHEPHERD, M.D., Kansas City

Information

Ownership: The Journal is a non-profit publication owned and published monthly by the Kansas Medical Society.

Subscription: A year's subscription to the Journal is included in membership in the Kansas Medical Society, with $2.00 of each member's dues apportioned to the Journal. Rates to others, except in foreign countries, $4.00 per year or 60c per copy.

Material: Scientific articles, editorials, and data of general interest are invited from all members. Articles are to be submitted on condition that they are contributed solely to this publication. A right is reserved to reject any material deemed unsatisfactory.

Manuscripts: Only manuscripts that are typewritten on one side, double spaced, and original copies can be accepted. Manuscripts will be returned upon request.

Advertising: All advertising contracts, and all copy from advertisers under contract are subject to approval of the editorial board. Copy should be received by the 20th of the month immediately preceding the month of publication.

Non-Responsibility: Although an effort is made to publish only accurate articles and legitimate advertisements, the Journal denies legal responsibility for any statements, opinions, or advertisements appearing under the names of contributors or concerns.

PAULINE FARRELL, Managing Editor
and Advertising Manager

OLIVER E. EBEL, Business Manager

THE KANSAS MEDICAL SOCIETY

President.....LUCIEN R. PYLE, M.D., Topeka
Past President....WARREN F. BERNSTORF, M.D., Winfield
President-Elect.....MURRAY C. EDDY, M.D., Hays
First Vice-President..JOHN M. PORTER, M.D., Concordia
Second Vice-President..CLYDE W. MILLER, M.D., Wichita
Constitutional Secretary..DALE D. VERMILLION, M.D., Goodland
Treasurer.....JOHN L. LATTIMORE, M.D., Topeka
AMA Delegate 1953-1954, LAURENCE S. NELSON, M.D., Salina
AMA Delegate 1954-1955, GEORGE F. GSELL, M.D., Wichita

Councilors and Councilor Districts

First District.....W. L. ANDERSON, M.D., Atchison
Second District .....H. P. JONES, M.D., Lawrence
Third District....CHARLES E. VESTLE, M.D., Humboldt
Fourth District.....F. C. TAGGART, M.D., Topeka
Fifth District.....HAROLD M. GLOVER, M.D., Newton
Sixth District.....NORTON L. FRANCIS, M.D., Wichita
Seventh District..SEVERT A. ANDERSON, M.D., Clay Center
Eighth District.....A. E. RUEB, M.D., Salina
Ninth District.....JAMES L. JENSON, M.D., Colby
Tenth District.....L. W. REYNOLDS, M.D., Hays
Eleventh District.....C. V. BLACK, M.D., Pratt
Twelfth District.....R. G. KLEIN, M.D., Dodge City

OLIVER E. EBEL, Executive Secretary
RUEBEN M. DALBEC, Executive Assistant

Executive Office: 315 West Fourth Street, Topeka

UNIVERSITY OF MARYLAND
LIBRARY
SCHOOL OF MEDICINE

44277

THE JOURNAL
of the
KANSAS MEDICAL SOCIETY
Copyright, 1954, by The Kansas Medical Society

Volume LV JANUARY, 1954 No. 1

The Treatment of Diabetic Acidosis and Diabetic Coma

Edward Tolstoi, M.D.
New York, New York

There is no one accepted technique for the treatment of diabetic acidosis and coma at present. Therefore, I can only discuss the method I have used and with which I have had the greatest experience. It is not presented as the most perfect plan of treatment, but as a plan that has given us satisfactory results. It is simple, safe, effective and does not require a specialist. No doubt there are many other methods, differing in details though not much in principle, which might have been just as efficacious in our hands. But, since I have had insufficient experience with other techniques, I shall describe ours in detail, and in passing make some comment about other methods.

For clarity I shall present the subject under the following headings: 1. Definition; 2. Presentation of cases and their management; 3. Why ketoacidosis develops; 4. How ketoacidosis develops; 5. A discussion of the accepted and controversial principles and procedures of treatment.

1. DEFINITION

A. *Diabetic Acidosis:* This is a clinical picture in which a patient with diabetes develops increased thirst, frequency, weakness, acetone breath, nausea, vomiting, abdominal and chest pains, soft eye balls, as well as other evidences of dehydration and air hunger (Kussmal breathing). The laboratory studies will reveal a glycosuria, ketonuria, hyperglycemia, leukocytosis and a low carbon dioxide combining power. However, the patient is conscious.

B. *Diabetic Coma:* When a patient with diabetic acidosis loses consciousness, then and only then is the diagnosis of diabetic coma made. Such a patient may be semi-conscious; that is, he will respond to stimuli, but may be unable to take fluids or answer questions, or, he may be unconscious so that he does not even respond to stimuli. I agree with Owens[1] that the term diabetic coma should be used only for those patients in ketosis whose mental state has become one of unconsciousness. It is wholly a clinical diagnosis. True, the carbon dioxide combining power is usually low. This indicates the degree of the acidosis only, but certainly not the mental status of the patient.

2. PRESENTATION OF CASES AND THEIR MANAGEMENT

A. *The Mild Case.* Often a trained diabetic patient under observation will carry on, feel well, hold his weight, and be free from symptoms, and though he has a glycosuria he will not show acetone in the urine. At times, during a slight upper respiratory infection, a gastrointestinal upset, or an extreme sunburn, or for no apparent reason, his thirst will increase, the urinary output rises, and acetone is found in the urine. The qualitative reading of the reaction may be 1 to 4 plus. Such a patient is not hospitalized. He is usually familiar with the tests for acetone and sugar and can follow his treatment at home. He is then told to follow these directions:

From the Department of Medicine, New York Hospital and Cornell University Medical College, New York City.
Associate Professor of Clinical Medicine, Cornell University Medical College; Attending Physician and Chief, Diabetic Clinic, New York Hospital; Consulting Physician, New York Infirmary for Women and Children, and Norwalk Hospital, Norwalk, Connecticut.

1. Test your specimens, every time you void, for sugar and acetone.

2. If acetone is present, take regular insulin in amounts depending upon the quantity of sugar found in the urine. That is, if the test if yellow, brown or orange (4 plus) take 25 units: if the test is green with yellow sediment, take 15 units: if the urine is blue or sugar free, take orange juice.

3. Drink all you can—water, milk, fruit juices, ginger ale.

4. Take salty broths as often as desired.

5. Continue this routine until 3 consecutive specimens are acetone free. If acetone persists and you feel nauseous or vomit, come to the hospital at once.

By means of this home treatment the ketosis clears rapidly and as the underlying cause improves, the patient can resume his former regimen. In those in whom the ketosis appears without apparent cause, I have learned that an increase of their daily dose of insulin remedied the situation.

B. *The Moderately Severe and Severe Case.* Before presenting a description of this group, I want to state categorically that every newly diagnosed diabetic with ketosis, regardless of its severity, should be hospitalized, chiefly because of lack of training. However, I must state that because of hospital overcrowding I have treated some of these cases at home. Here the help of a nurse or a member of the family was urgently needed. I taught them the routine of testing, left definite written instructions, kept in touch by telephone at hourly intervals, and made calls as often as I could. There were no dire consequences.

Now, the moderately severe case developing ketoacidosis and coma may reveal this pattern. The patient develops a sore throat or some other infection. His temperature rises, he develops malaise, and he has no desire for food. Since he decided not to eat, he sees no necessity for taking insulin as he fears a reaction. He may not eat for a day, two days or more, subsisting on fruit juices and other fluids and still reasoning, though fallaciously, that insulin under these circumstances is unnecessary. Soon he observes an increase in thirst and notes some frequency. He then develops aching legs, weakness, restlessness, and, perhaps, some somnolence. He carries on. Soon there appear epigastric pain, nausea, and vomiting. His heart beats rapidly, and it becomes difficult for him to breathe. He or some member of the family becomes alarmed, and either a physician is called or the patient is taken to the hospital. Often no medical aid is sought until the patient is or has been unconscious for many hours. When such a patient is admitted to the hospital, the infection will be treated by any of the modern chemotherapeutic or antibiotic agents, and his diabetic acidosis, coma, or both will be treated as follows:

1. If the patient is unconscious, insert a catheter into the bladder and clamp it. This will facilitate getting samples of urine for analysis.

2. Give 25 units of regular or crystalline insulin subcutaneously every half hour until the clinical symptoms of ketosis begin to disappear and the urine becomes acetone free.

3. If the patient can take and retain fluids, give 2 or 3 glassfuls (200 cc.) of fruit juice every half hour to one hour. Salty broths and other liquids are given as frequently as the patient desires.

4. If fluids cannot be taken by mouth, give 1000 cc. of 5 per cent glucose in saline at once, rapidly. The infusion is then continued at a slower rate until the oral route can be used. In connection with any

TABLE 1

		BLOOD		URINE			
TIME	INSULIN	CO_2	SUGAR	SUGAR	ACETONE	DIACETIC	
8:00	25 units	19	325	4 plus	4 plus	4 plus	1000 cc 10% G. in Sal.
8:30	25 "			4 "	4 "	4 "	400 cc water
9:00	25 "			4 "	4 "	4 "	200 cc O.J.
9:30	25 "			4 "	4 "	4 "	500 cc N/6 lactate
10:00	25 "			4 "	4 "	2 "	200 cc water
10:30	25 "			4 "	4 "	0	200 cc water
11:00	25 "			4 "	4 "	0	1000 cc 10% G. in Sal.
11:30	25 "			4 "	4 "	0	400 cc water
12:00	25 "			4 "	4 "	0	200 cc water
12:30	25 "			4 "	4 "	0	200 cc O.J.
1:00	25 "			4 "	4 "	0	400 cc water
1:30	25 "			4 "	4 "	0	400 cc water
2:00	25 "			4 "	4 "	0	1000 cc 10% G. in Sal.
2:30	25 "			4 "	4 "	0	100 cc O.J.
3:00	25 "			4 "	3 "	0	200 cc water
3:30	25 "			4 "	3 "	0	200 cc water
4:00	25 "			4 "	2 "	0	100 cc grape J.
5:00	25 "			4 "	±	0	500 cc 10% G. in Sal.
6:00	25 "			4 "	0	0	200 cc water
7:00	25 "			4 "	0	0	

Patient had diabetes for 12 years. Was taking R20—0—P25. Well until one month ago when he began losing weight; polyuria and loss of appetite. Three days ago was nauseated and did not take insulin. For 2 days before admission vomited all food, became drowsy, and, when admitted, vomited a small quantity of blood.

P.E. P120 R40 BP 105/50 T37
Semi-stuporous, Kussmal, acetone in breath, eye balls soft. Pharynx red.
HBO 16; RBC 5.1 : WBC 29,800; BUN 86
Acetone free in 9 hours.

continuous intravenous therapy, great care must be exercised to prevent overloading the right heart. This hazard is present in the older diabetic with arteriosclerosis and the young cardiac.

5. After the urine has become free from acetone bodies continue giving 25 units of insulin and 2 glasses of orange juice every two hours for four or more doses.

Of 165 cases of diabetic acidosis and coma, so treated, there were 19 deaths of which only 6 were due to the acidosis per se, and of these one was highly insulin resistant.

3. WHY DOES KETOACIDOSIS DEVELOP?

The factors responsible for the development of diabetic acidosis and coma are: 1. failure to take insulin; 2. infection; 3. combination of the two. In Joslin's experience, "The commonest cause of diabetic acidosis continues to be laxity of treatment, consisting of dietary indiscretions or neglect to take an adequate amount of insulin, or both." These factors were the cause of 53 per cent of a group of cases, infection being accountable for only 23 per cent. In my experience, infection and omission of insulin were the commonest causes for the precipitation of ketoacidosis and coma.

The dietary breaks were not as impressive, and after an analysis of 27 of our patients who did not adhere to a diet, we found that only 2 were eating more while 25 ate less than their prescription called for. In a group of 220 instances of diabetic acidosis and coma, Beardwood and Rouse[2] state, "Only nine can be listed as breaking diet." Though there is a difference of opinion concerning the "breaking of diet" as a factor in the development of diabetic ketoacidosis, it can be categorically stated that the most important cause is the omission of insulin, or a relative insufficiency of it, as during an infection its efficacy is considerably reduced. Consequently, a unitage which may have been sufficient during health to maintain the urine free from ketones, becomes inadequate during an infection, and ketosis ensues.

4. HOW KETOACIDOSIS DEVELOPS

At this point it is of interest to inquire how the diabetic develops ketoacidosis and coma. As you know, the overabundance of acetone bodies, acetone, diacetic, and beta hydroxbyteric acids, in the blood and tissues causes an acidosis. This is brought about by a depletion of base. It was once thought that these acetone bodies were abnormal and products of fat metabolism. This hypothesis is not tenable today.

The experimental studies of many investigators have established the following facts:

A. Ketone bodies are normal intermediates of fatty acid catabolism in the liver—the only organ which manufactures them in significant quantities. B. Ketone bodies so made in the liver are secreted into the blood stream where they are utilized by the peripheral tissues. This occurs in both normal and diabetic subjects, whether or not insulin or carbohydrate is available. C. Ketone bodies will not cause acidosis as long as their production does not exceed their utilization. However, when they are produced at a greater rate than they can be utilized by muscle and other tissues, they must of necessity accumulate, and it is this accumulation in the blood and tissues, due to over-production, that starts the vicious cycle of acidosis, depletion of base, dehydration, and coma.

The next query is, "Why does the liver overproduce these ketone bodies?" The obvious reply is that the liver is using for its energy the substance from which these bodies are derived—namely, fatty acids. Stadie's[3] experimental studies led him to the conclusion that when the demand for fat calories exceeds about 2.5 gms. per kilogram per day, the production of ketones exceeds utilization resulting in ketonuria, and, if unchecked, leads to ketoacidosis and coma.

How can we prevent the liver from subsisting on fatty acids? By making carbohydrate available as liver glycogen. Liver glycogen has been termed the greatest factor of safety against ketogenesis. Here the hypothesis has been proposed by Mirsky[4] and supported by fine experimental evidence that a decrease of the liver glycogen is the most likely stimulus to ketone formation. This is quite logical, for, as the available carbohydrate is lowered, the liver will metabolize protein and fat. And as the combustion of fat reaches the figure of 2.5 gms. per kilogram per day, as postulated by Stadie, the production of ketones will be accelerated to a point surpassing utilization with a sequential ketosis.

If, therefore, we can offer the liver carbohydrate (glycogen) instead of fat, will ketogenesis be decreased? Evidence answers this query rather conclusively. A rapid drop in ketonemia and ketonuria was observed by Mirsky[4] in depancreatized dogs following the administration of glucose intravenously. Root and Carpenter[5] also reported a drop in blood ketones from 70 to 44 mg. in a diabetic patient 3 hours after 50 gms. of glucose were given by vein. It is obvious that glucose alone can decrease acetone formation in both the diabetic animal and the diabetic patient, but, to suppress ketogenesis, insulin is also needed. It has been shown that the glycogen content of the diabetic liver increases more markedly when insulin is also used, and that the glucose so stored is not so rapidly dissipated, thus offering the liver carbohydrate rather than fat for its own metabolic needs.

It is obvious from what I have said that the diabetic patient will develop ketosis and coma if the liver catabolizes fatty acids in abnormal quantities.

This the liver will do if glycogen is not available for its energy requirements. And, in the diabetic the reduced glycogen stores are further lowered by omission of insulin, infection, or any other condition which reduces the potency of insulin.

5. PRINCIPLES AND PROCEDURES OF TREATMENT

Each group employing the technique it developed for the treatment of diabetic acidosis insists that all steps of treatment are based on sound physiologic grounds. Mortality statistics are produced to support the thesis that such treatment is most satisfactory, and, as additional support explanations are offered, why the death rate is higher elsewhere. Such comparative data are neither satisfactory nor valid because of the multiplicity of factors which determine the prognosis in each case.

It is generally agreed that the results are dependent on: A. the duration of unconsciousness; B. the age of the patient; C. the presence of serious medical complications.

If similar cases could be treated by different methods, the superiority of one method over another could be determined. However, such an ideally controlled clinical experiment is always beyond hope. Owens[1] has stated that even the so-called "best therapy" in the unconscious diabetic is highly unsatisfactory while the duration of acidosis without unconsciousness is of considerably less importance. All agree that for best results early treatment is imperative.

The most commonly and frequently employed measures in the treatment of diabetic acidosis and coma are:

1. insulin
2. fluids and salt
3. blood analysis
4. glucose
5. potassium
6. adjuvants
   a. N/6 molar lactate
   b. transfusions
   c. alkalis
   d. lavage
   e. enemas
   f. stimulants

Everyone agrees that insulin is most urgently needed as diabetic acidosis and coma are the end results of a relative or absolute insulin insufficiency. Everyone, therefore, prescribes insulin—the difference is a quantitative one. The dosages recommended by various workers have varied from 25 to 400 units as the initial dose. Whenever the smaller doses are given, they are repeated at one-half to hourly intervals until improvement as judged by clinical and laboratory data is apparent. Olmstead[6] gives 100 units at once, and, if there is no drop in the blood sugar after 1½ hours, another 100 units are given. Joslin's group of 141 cases received an average of 206 units during the first three hours.

There are still other variations, and the fact that good to excellent results have been obtained by all methods suggests that insulin is important, but that one dose or its frequency of administration is not superior to another dose given more or less frequently. We have given 25 units, a small dose, at frequent intervals of 30 minutes. This plan, though empirical, has been satisfactory. That does not mean that giving 50, 75, or 100 units is not good therapy, even though the outcome in a series of cases may be poor.

Owens[1] used 50 units of insulin every half hour at the start, but he reports a high mortality. Of 92 patients admitted, 49 were unconscious with an average of 16 hours of coma. Among this group of 49, there was a death rate of 73.5 per cent. Does that mean that Owens' technique is inferior to ours, or that some one bungled the therapy? Not at all. His work is done in a general hospital with an active ambulance service, and he is called upon to treat more severe and more neglected cases than we do.

I decry the statement that some one has blundered if death occurs as a result of diabetic acidosis or coma. Many patients die in spite of generally accepted "superior" therapy, and no satisfactory explanation can be offered as to why death occurred. Schacter, Weisel and Cohn[7] suggest that peripheral vascular failure causes such deaths. Danowski, Winkler and Peters[8] state that the profound loss of salt and water may be the causal factors of such failure. These hypotheses may be tenable as the patient in ketosis with shock has a decreased blood volume, a hemoconcentration, a hypochloremia, a hyperpotassemia, and a falling blood pressure. This may be the picture terminally. However, I have observed comatose diabetics whose blood pressure was maintained, whose chemical picture was corrected, and in whom no clinical evidence of peripheral failure was apparent. The therapy was considered adequate, and yet such patients never regained consciousness.

The use of salt and fluids to replace the loss of base and water is so universal that no comment is needed. Often sixth-molar lactate was also used, and transfusions were given only as indicated. We do not rely upon the level of the blood sugar, nor carbon dioxide combining power, as a guide of treatment. In our hospital and probably in others, the results of the blood examination may not be available for one to three hours, at which time the data are of academic interest. Our principal laboratory guide is the examination of urine for sugar and acetone at ½ hourly intervals, and we continue the insulin dosage of 25 units as long as acetone is present. The test is reliable, rapid and therefore practical.

The question of whether or not glucose should be used in the treatment of diabetic acidosis and coma has been argued with much passion and emotion. Many statements concerning the deleterious effects of glucose have been based on circumstances that were merely fortuitous. Our experience with glucose has not been unfavorable. As I have indicated above, we have used it as a 5 per cent and occasionally as a 10 per cent solution intravenously. The median given in a 24-hour period was 112 grams. When the amount given by mouth was added to that, the median amount rose to a total of 312 grams for the first 24 hours, with a minimum of 50 grams and a maximum of 800 grams.

It has been stated by Root[9] and his associates that glucose in doses of 50 or more grams is harmful because: 1. It obscures the actual blood sugar values, thus eliminating a guide for insulin dosage; 2. It may produce liver damage; 3. It neutralizes the action of insulin; 4. The hyperglycemia thus produced is harmful to the pancreas; 5. It may provoke anuria; 6. Such large doses are not necessary as the body can metabolize only 5 to 10 gms. per hour.

These statements are made by respected leaders in the field with such conviction that the physician whose opportunity to treat diabetic acidosis is limited, accepts them as facts. It is desirable to appraise them and see what evidence is available to support them.

That the administration of glucose obscures the actual blood sugar is a fact, and I have no objection if one wishes to use the blood sugar as a therapeutic guide. I maintain, however, that it is excessive ketonemia and not hyperglycemia which is the underlying cause of diabetic acidosis and coma, and, therefore, ketone bodies in the urine are more accurate guides of therapy. Insulin should be continued frequently until the ketone bodies vanish, and then it is no problem if one wishes to treat the hyperglycemia.

Astwood, Flynn, and Krayer[10] have shown that liver damage resulted in dogs receiving glucose by vein at a rate exceeding 40 gms. per square meter per hour. They also demonstrated that dogs indefinitely tolerated 12 gms. per square meter per hour. These experiments have been offered as evidence that glucose may produce liver damage. Such reasoning is fallacious. First, excessive quantities of any innocuous agent, even water, will prove toxic, and while a dog may not be able to tolerate large quantities of glucose, that does not necessarily indicate that man cannot do so. Recently we had an opportunity to observe a patient in whom large quantities of glucose were not hepatotoxic. Following a transfusion, a young woman developed a kidney shutdown. To facilitate diuresis, large quantities of glucose were given by vein. The data is shown in Table 2.

This patient, as you see, received from 300 to 870 gms. of glucose daily by vein. There was no glycosuria. Most of her blood sugars were normal, and one is led to the conclusion that glucose so given was metabolized at a rate of 12.5 to 36 gms. per hour. Histologically the liver showed no abnormalities. Rabinowitch, Fowler and Bensley[11] have shown that the diabetic with the aid of insulin can also utilize more than 5 to 10 gms. of glucose per hour, as shown in the table.

TABLE 3

HOURS	32	48	43
GLUCOSE INTAKE	1020	780	835
EXCRETED	10	32	63
UTILIZED	1010	748	772
GMS/HOUR	31.5	15.5	18.0

It is true that Root and Carpenter[5] used a different approach in determining how much glucose was metabolized. However, other data as well as ours are clear. It is obvious that if the glucose was not excreted, it must have been used by the body.

The "neutralization" of insulin by glucose is questionable. Perhaps that is not what was meant, but the choice of the word "neutralize" was not the most desirable one.

The interaction of insulin and glucose is not the same as the neutralization of an acid by an alkali. Insulin favors the accumulation of glycogen, but its

TABLE 2

DATE	FLUID INTAKE	URINE OUTPUT	GMS. GLU. 24/HRS.	UREA N.	BLOOD				ICTERIC INDEX
					SUGAR	CO_2	CHLORIDES	SERUM P.	
2/11	6350	170	870	65	223	62	479	4.4	5
2/12	2700	73	490	65	133	60	469	5.5	—
2/13	3795	21	760	69	190	49	446	5.5	11
2/14	2360	30	300	73	85	57	429	5.5	
2/15	3930	47	700	85	89	51	423	5.1	
2/16	3900	37	400	—	—	—	—	—	
2/17	2800	105	300	—	—	—	—	—	
2/18	4970	108	425	89	77	45	399	4.8	5
2/19	3815	908	400	100	79	41	376	—	
2/20	3885	1930	300	104	—	40	385	5.0	
2/21	4330	3150	500	111	85	40	396	5.1	
2/22	4200	3850	500	102		45	421	—	
2/23	0	4200	400	96		37	424	5.4	

mode of action is not yet understood. Its action has been compared to an enzyme subtrate reaction, and, whether or not an excess of glucose is present, a certain amount will be acted upon by the quantity of insulin available. The beneficial effect of the insulin is not nullified by excessive amounts of glucose.

The harmfulness of excessive quantities of glucose to the pancreas, in the treatment of diabetic acidosis and coma, is highly speculative. My own observations on postmortem material failed to reveal histological changes in the pancreas. This is not an unusual observation as Root also quotes two fatal cases which were treated with glucose in addition to insulin and whose blood sugars were 475 and 667 mgm. per cent, and yet the autopsies were negative. It seems to me that there is altogether too much condemnation of glucose in the treatment of diabetic acidosis and coma. This indictment is not based on sound evidence.

I doubt whether the use or non-use of glucose is the crucial issue. I am sure that suitable cases will recover both with and without the use of glucose as long as adequate doses of insulin, salt, and fluids are used. The case in which there has been a long period of unconsciousness will probably die in spite of the so-called very best treatment. Personally, as I stated, with frequent small doses of insulin and the use of glucose, I have found the results good. The addition of glucose gives an added feeling of security that I will not throw my patients into the hypoglycemic phase, thus putting an additional burden upon a circulatory system already under considerable stress.

Potassium. In 1946 Holler[12] noted that a patient, while recovering from diabetic coma, developed paralysis and at the same time exhibited a low serum potassium. The administration of a 2 per cent solution of potassium chloride intravenously resulted in a dramatic recovery from the paralysis. It was quite natural, therefore, that potassium salts began to be used rather freely and generally in the treatment of diabetic acidosis and coma. As experience with potassium salts as a component of treatment increased, many physicians urged that their use be most judicious because of toxic reactions. This is not the place to enter into a detailed discussion of potassium metabolism. Briefly, it is accepted by those who have had experience with potassium, that the greatest danger of a hypopotassemia is during the recovery phase, after the patient's acetone has cleared because of large doses of insulin, fluids, salt, and glucose. At this stage the patient is usually conscious, not nauseous, and is able to retain fluids. Consequently, the safest and best way to replenish the lost potassium content is the administration of substances rich in potassium. Orange juice, grapefruit, and grape juice are ideal. If more potassium is desired, it can be administered as the chloride or citrate as 1.0 gm. every 6 hours.

However, if after diligent and adequate therapy, the patient is still unconscious and one wishes to administer potassium, a solution can be prepared from commercially available ampules containing 1.5 gms. per 10 cc. Usually such a 10 cc. ampule is added to 500 cc. of 5 per cent glucose and permitted to flow into a vein at the rate of 60 to 75 drops per minute.

Prior to the intravenous use of potassium, one must ascertain that there is a hypopotassemia and that the kidneys are functioning well with a good urinary output. Furthermore, the serum potassium levels should be determined by the flame photometer and also electrocardiograms. While the electrocardiographic tracing usually reflects the serum potassium concentration, at times the correlation is poor. I have not used potassium routinely by vein as it is a hazard. It should be employed sparingly, judiciously, and only by those whose facilities for analyses are good and whose experience has been extensive. The oral route is safer and certainly less troublesome.

SUMMARY

1. Patient with diabetic acidosis or diabetic coma can be successfully treated with small and frequently repeated doses of regular insulin—25 units every half hour—replacement of water, salt, and glucose by mouth, vein, or both.

2. The administration of glucose by vein or orally is beneficial and not harmful. Data are presented to support this thesis.

3. Potassium salts are not used routinely in the treatment of diabetic ketosis and coma. This does not mean that they have no place in therapy. They have a definite value, but there are hazards associated with their use. Both of these points have been discussed.

4. It is unnecessary to follow the blood sugar and the carbon dioxide combining power as guides of therapy. Our experience has shown that clinical response and disappearance of ketone bodies are excellent guides.

5. The earlier the treatment is begun, the more favorable is the prognosis, and, exclusive of medical complications, it can be stated categorically that the longer the patient remains unconscious, the less favorable are his chances for recovery.

BIBLIOGRAPHY

1. Owens, L. B.: The treatment of diabetic coma, Cincinnati Jour. of Med. 25:286, 1944.
2. Beardwood, J. T., and Rouse, G. P.: Diabetic acidosis, J.A.M.A. 117:1701, 1941.
3. Stadie, William C.: Fat metabolism in diabetes mellitus, Ann. Int. Med. 15:783, 1941.
4. Mirsky, I. A.: The etiology of diabetic acidosis, Proc. Am. Diabetes Assoc. 1:51, 1941.
5. Root, H. F., and Carpenter, T. M.: The effect of glucose administration in diabetic acidosis, Am. J. Med. Sci. 206:234, 1943.
6. Olmstead, W. H.: Diabetic Acidosis, Med. Clinics N. A. 26:375, 1922.

7. Schecter, A. A.; Weisel, B. H., and Cohn, C.: Peripheral circulatory failure and diabetic acidosis and its relation to treatment, Am. J. Med. Sci. 202:364, 1941.
8. Danowski. R. S.; Winkler, A. W., and Peters, J. P.: Salt depletion, peripheral vascular collapse and the treatment of diabetic acidosis, Yale Jour. Biol. & Med. 18:405, 1946.
9. Root, H. F.: The use of insulin and abuse of glucose in the treatment of diabetic coma, J.A.M.A. 127:557, 1945.
10. Astwood, E. B.; Flynn, J. M., and Krayer, P.: Effect of continuous intravenous infusion of glucose in normal dogs, J. Clin. Investigation 21:621, 1942.
11. Rabinowich, I. M.; Fowler, A. F., and Bensley, E.: The use of protamine insulin in diabetic coma, Canad. M.A.J. 37:105, 1937.
12. Holler, J. W.: Potassium deficiency occurring during the the treatment of diabetic acidosis, J.A.M.A. 131:1186, 1946.

Management of Acute Laryngotracheobronchitis

Harold L. Hickey, M.D.

Denver, Colorado

In a recent symposium at a national meeting, croup and laryngotracheobronchitis were listed under the same title, although I believe the intent was to give consideration to the latter disease. Croup is a relatively benign disease, and its management should prove no problem. Confusion of the two conditions may be due to the fact that acute infective laryngotracheobronchitis has sometimes been called nonmembranous croup to distinguish it from diphtheria, formerly known as membranous croup.

The condition under consideration may be defined according to Jackson[1] as "an acute inflammation of the upper and lower air passages with specific clinical features but not always attributable to the same infective agent." The disease in its incipiency is frequently mistaken for croup, and it may be confused with laryngeal diphtheria, but its fulminating character and severity should change this opinion; resultant deaths, often diagnosed as pneumonia or bronchopneumonia, are in reality the terminal phases of unrecognized acute laryngotracheobronchitis.

To set the stage for our discussion, I should like to point out a few etiologic and diagnostic features because I so firmly believe that they have an important bearing on treatment. The chief predisposing cause is age, the greatest incidence being infancy to four years, with the highest mortality under two years. Factors contributing to the clinical picture in relation to age are: (1) Lack of adequate immunity in a child who has never had an acute respiratory infection. (2) Anatomic reasons: in the early years of life, there is much more loose areolar tissue in the larynx, especially in the conus elasticus. The triangular aperture of the infant larynx[4] is 7 mm. in anteroposterior direction by 4 mm. in width across the posterior commissure. The glottic chink has an area of approximately 14 square mm. An edema of 1 mm. of the mucosal surfaces of this glottic chink will reduce the airway to 5 square mm. Also the lumen of the airway below the larynx is relatively small. Thus, an exudate 1 mm. in thickness on the lining wall will completely occlude a 2 mm. bronchus while the same exudate in a 4 mm. bronchus would leave a 2 mm. lumen. (3) Physiologic factors: the bechic blast (force of cough) in a baby is about 1/10 that of an adult, and the child is unable to expel sticky secretions from small tubes. The cough reflex may be obtunded or absent because of toxemia or ill advised drugs.

In addition to the above, a careful history will commonly reveal contact infections of the influenzal type with tracheobronchitis, in older members of the family. I can personally testify to a reverse twist of this situation when, in 1936, I treated a serious case of laryngotracheobronchitis which required bronchoscopy and tracheotomy. As a result, in 48 hours I contracted a severe tracheobronchitis which progressed to bronchopneumonia. As an adult I survived, but the child succumbed.

The hairline distinction between obstructive nondiphtheritic croup and infective laryngotracheobronchitis is not important; both usually present the same characteristics in typical cases: toxemia, edema of the larynx, and thick viscid obstructive, often crusting, non-membranous exudate in the tracheobronchial tree.

Gilbert[2] has given us a useful classification according to the pathologic changes present and often observable with the laryngoscope or bronchoscope:

1. Acute catarrhal laryngotracheitis in which the laryngeal and upper tracheal mucosa is reddened and velvety, secretions are mucoid and easily aspirated, and the cords freely movable; clinical symptoms of hoarseness, brassy cough, and obstruction are comparatively mild.

2. Supraglottic edematous obstructive laryngitis in which the epiglottis is enormously swollen and beefy red, edema involves the aryepiglottic folds and arytenoids so that one or both cords are fixed. The picture of obstructive dyspnea is rapidly progressive. The voice is guttural, wavering, and described as having a "hot potato" quality. The patient assumes an up-

Presented before the Eye, Ear, Nose and Throat Section of the Kansas Medical Society, May 5, 1953.

right position with chin forward and mouth open, and respiratory distress increases in the recumbent position.

3. Subglottic obstructive laryngitis and tracheitis further subdivided into (a) the exudative or inspissated type, and (b) the edematous type. In the exudative type, laryngoscopic inspection reveals vocal cords slightly separated by moist exudate or crusts; after aspiration the cords move freely. A congested nonedematous mucosa is seen in the subglottic area, and dyspnea is relieved, if only temporarily. In the edematous type, supraglottic structures are normal, the cords are slightly mobile or fixed in the midline, and suction does not increase their mobility; the subglottic edema narrows the airway and prevents normal abduction of the true cords during inspiration. Hence, aspiration of secretions does not relieve the dyspnea in this type.

4. Acute obstructive laryngotracheobronchitis, according to Gilbert[2] is a term applicable to only a small group of cases (8 per cent in his series of 2,550 cases) in which subglottic edematous laryngitis with fixed cords is combined with edematous tracheobronchitis and a mucopurulent exudate or gummy material which extends into the smallest bronchial radicles. Aspiration, tracheotomy, or even bronchoscopy may not relieve these patients who are usually under 1 or 2 years of age, and the mortality rate remains about 70 per cent. In my opinion, it is in this type of patient that the recent combination of a wetting agent as advised by Gorman,[3] along with cold humidification, offers some additional hope by liquefaction of the almost solid exudate and making possible catheter aspiration through the tracheotomy tube.

As with all classifications, this one has its limitations; various types may not be sharply differentiated or may merge one with the other. Any intelligent consideration of this disease entity adds up to the inevitable conclusion that we are dealing with a serious illness in the younger age group.

In medical management of the disease, the young patient requires prompt judgment when first seen. The notoriously rapid onset, stridulous croupy respirations, high fever, and extreme toxicity give warning, but obstruction to breathing decides that hospitalization must be immediate.

The most important therapeutic indication is placement of the patient in a room saturated with moisture. Steam is the traditional method and in some cases is effective. However, the steamy room with temperature at 90°, the ceiling and walls dripping, a constantly moist bed, are overbearing and can be harmful. A nurse is able to work under such conditions only a few hours. The same situation within a crib-tent embarrasses the patient. The larger the tent, the easier the handling, and the patient has the feeling of adequate space for breathing.

Cool moist air is soothing to the mucous membranes and helps to lower body temperature and metabolic rate; hence, there is minimum oxygen consumption. Loss of body fluids is also much less.

Relative humidity of 70 to 90 per cent with oxygen concentration of 40 per cent at a room temperature of 65° to 75° can be maintained by various methods. With infants, a Burgess box serving as a croup tent into which moisture combined with a wetting agent is nebulized, is quite satisfactory. The wetting agent, sodium lauryl sulfate (Duponel) is cheap and nontoxic, and humidity can be noticeably increased, up to 30 per cent, by its use. Oxygen from a cylinder at the rate of 4 to 6 litres per minute can be used as the nebulizing agent. Several other means have been devised, but our judgment favors the Walton centrifugal humidifier, which is safe, durable, and requires no mechanical tricks to operate. It can be used in conjunction with the oxygen tent if desired.

General measures for the comfort and nutrition of the patient should be so arranged to cause minimum disturbance. Postural drainage as advocated by Galloway[10] is of great advantage. Adequate fluid intake is essential; it should be encouraged by mouth when possible and supplemented parenterally if needed. Since restlessness is an important diagnostic sign of increasing obstruction, sedatives should not be used. Opiates and atropine are strongly contraindicated.

Early antibacterial treatment has been a potent factor in the decrease of morbidity. Culture and sensitivity studies would be desirable if time permitted, but the urgent need for active treatment precludes such study in most cases. Hemolytic streptococcus and staphylococcus and hemophilus influenzae are the most constant causative organisms.

Penicillin becomes the first choice of antibiotics, and it must be given in full dosage. Streptomycin, because of its selective effect on influenzal infections, may be combined with penicillin. Aureomycin, terramycin, and the sulfonamides may have value in some cases.

Before discussing the actual surgical procedures available for treatment, it should be useful to outline some of the indications for surgery. The medical management, as outlined, may obviate the necessity for surgical intervention. The important indications then would seem to be:

1. Failure to improve under medical care with watchful and alert observation.

2. Increase in the signs of obstructive dyspnea, such as stridorous breathing, air hunger, retraction at the suprasternal notch and epigastric (xyphoid) region, also supraclavicular, infraclavicular, and in-

tercostal retraction. True cyanosis is a late symptom, and to wait for its appearance will be too late. Pale cyanosis described as an ashen gray color is an important sign of serious obstruction.

3. Restlessness as exhibited by a sudden start on awaking when a worn out child has momentarily lapsed into fitful sleep and suddenly discovers that his airway is obstructed.

4. Lessening or disappearance of cough because the reflex has been obtunded by toxemia or the use of sedative or mucosal drying drugs.

5. Sudden hyperpyrexia is an unfavorable diagnostic and prognostic sign.

Having decided that surgical care is needed, what are the possible methods available? Direct laryngoscopy may be used for augmenting the diagnosis, for aspiration of secretions and crusts, but is most important as a preliminary to introduction of the bronchoscope for deeper aspiration and to provide an immediate airway. Hollinger and Johnston[4] have pointed out that instrumentation in an infant's larynx may precipitate complete respiratory obstruction by laryngospasm incident to introduction of the laryngoscope. One must therefore be prepared to establish an airway by Mosher life-saver, intratracheal catheter, or bronchoscope. This opinion is shared by Everett[9] who states, "It is difficult to imagine a completely harmless laryngoscopy."

Formerly intubation was used either by the indirect O'Dwyer method or the direct method of Mosher, but this was probably a carry-over from the days of widely prevalent laryngeal diphtheria. It has been largely discarded because the presence of an intubation tube requires the constant attendance of a physician capable of immediately replacing a coughed out or occluded tube, and it permits no aspiration of secretion from the trachea and lower respiratory tract. Repeated bronchoscopy is not advisable as it encourages laryngeal edema and usually gives only transient help.

The operation of choice in the acutely obstructed patient is tracheotomy,[5] and while a competent trained laryngologist should be familiar with the technique, complications, and aftercare of tracheotomy, there may be occasions when those less skilled are called upon to perform the operation.

When tracheotomy is anticipated as an orderly procedure, as much preparation as possible should be made in advance so that unnecessary confusion will not add to the apprehension of a dyspneic patient. Instruments should be sterilized, suction machine available, anesthetic syringes, solutions, etc., ready for use. Position of the patient is important, with a sandbag under the shoulders (not the head or neck) so that the head is extended; the chin must be in the midline. Extremities and the body must be carefully restrained before draping. Introduction of a Mosher life-saver or small bronchoscope facilitates aspiration, maintenance of an airway, and oxygen insufflation. Local anesthesia is introduced by a succession of intradermal wheals in the midline from the suprasternal to the thyroid notch. A few deeper injections may be made in the midline but are not necessary laterally as the operation is advisedly limited to a midline procedure. Jackson[1] forbids general anesthesia under any circumstances, while a few authors, including Crowe,[6] sanction light ether anesthesia with oxygen through a bronchoscope or intratracheal catheter, thereby making the operation much less of a shock or struggle for an ill child. This can be discontinued as soon as the trachea is opened.

Midline incision through the skin and superficial tissues is made from the Adam's apple to the suprasternal notch, taking care not to cut through the perichondrium of the laryngeal cartilages. Hemostasis is secured to make a dry wound, as in any surgical operation, and the incision is deepened to the pretracheal fascia, identified by the ridges of the tracheal rings felt by the finger. The thyroid isthmus, if atrophic, may be retracted upward or downward; if sizable, it should be incised between two clamps introduced from above down and ligated lateral to the clamps. After exposing the trachea, a hook tenaculum is used to fix and slightly lift the trachea by passing it below and under the second ring in the midline.

Vertical incision of the cartilages of the 3rd and 4th rings is now made, with care that the knife does not go deep enough to cut the posterior tracheoesophageal party wall. Priest[7] injects a few drops of 5 per cent cocaine solution into the trachea a few moments before opening, but Jackson[1] objects to this procedure as the cocaine dulls the cough reflex which acts to expel secretions on opening the trachea. Removal of a semicircular section from each side of the vertical incision leaves a circular opening about the same size as the tracheotomy tube and may avoid pressure necrosis at a later time.

The edges of the incision are spread with a Trousseau dilator to allow catheter suction in the trachea and easy introduction of the proper size tracheotomy tube with obturator; the latter is commonly forced out by bechic blast on release, but, if not, it should be removed and replaced by the inner tracheotomic canula. The upper end of the wound may be closed by two sutures well away from the tube, if desired, but leaving the lower end of the wound unsutured makes less likely the unhappy complication of emphysema in the tissues of the neck or even in the mediastinum. This will be discussed more fully later.

A split gauze dressing placed under the shield

and around the canula of the tracheotomy tube can be replaced when soiled and encourages normal healing of the wound in the presence of serum from the tissues. The tapes from the two sides of the shield should be joined around the neck and tied in a square knot rather than a bow knot. Failure to fix the tube snugly in position may allow a tube to be extruded, and deaths have resulted from this avoidable accident.

Emergency tracheotomy may occasionally be imperative. A knife and a pair of trained hands are enough,[1] but full surgical equipment is better. No anesthesia is needed for a patient requiring a life-saving emergency tracheotomy. The Mosher life-saver, which can be introduced by touch and used in a home, may obviate the emergency and make possible an orderly operation. The same cardinal principles of incision in the midline, feeling for the tracheal rings, and opening of the trachea with a finger limited scalpel, govern the emergency operation.

In the aftercare of tracheotomy patients, it is trite to say that success depends largely upon good nursing. However, the surgeon must not relinquish his responsibility or supervision to see that the proper care is forthcoming. Since the patient has been temporarily deprived of voice and ability to cry, he must be watched day and night lest the newly provided airway become again obstructed. Moisture must be supplied, preferably by a mechanical cold vaporizer, so that room or crib temperature is not above 72°F. In Europe[4] during World War II, a significant reduction in cases of laryngotracheobronchitis was observed when fuel was scarce and houses were heatless during most of the winter months. Before the war, this infection was prevalent in the winter months. Transportation of a sick child from the heated surroundings at home through the cool night air to the hospital has shown a similar favorable effect.

As previously noted, under medical care the addition of a wetting agent, sodium lauryl sulfate (Duponel C), as reported by Gorman,[3] appears to be an advantage. One cc. dissolved in one gallon of water can raise the relative humidity of a small room from 65 to 90 within 30 minutes. The same agent in .1 per cent solution in warm normal saline may be instilled directly into the tracheotomy tube, followed by gentle catheter suction to assist in removing the dry secretions and crusts from the tracheobronchial tree. The inner tracheal canula should be changed frequently by nurses, and the outer tube should be inspected daily by the surgeon and changed as needed. A Trousseau dilator and duplicate tracheotomy tube should be at the bedside; in some cases it may become necessary to remove crusts by passing a small bronchoscope through the tracheotomy wound. The patient must be watched for signs of hypoxia.

Decanulation (removal of the tracheotomy tube) can usually be accomplished in 7 to 10 days, but this should not be attempted until the patient has been on trial for 24 to 48 hours with the tube corked. Since many of these small patients become panicky when the tube is removed, having learned to depend upon it for free breathing, it may be wise to substitute the next smaller size tracheotomy tube for 2 or 3 days before attempting decanulation. If the tracheotomy tube has been placed at the 3rd or 4th ring, decanulation is not usually difficult. On the contrary, an emergency "stab" operation performed near the cricoid may result in stenosis, a troublesome complication.

Tracheotomy may be subject to some serious or even fatal complications. Emphysema, by forcing of air through the tracheotomy wound into the fascial planes of the neck and thence to the mediastinum, should be suspected when hypoxia and dyspnea recur in the presence of a clear airway. This accident is favored by suturing the wound, especially the lower end, too close to the tube. Hollinger[8a] has described another mechanism. He states that mediastinal emphysema and pneumothorax following tracheotomy with a bronchoscope in place are due to too low a tracheotomy, caused by hyperextension of the head which draws the trachea out of the thorax.

The fact that serious complications can and do occur is attested by Everett[9] in an analysis of acute laryngotracheobronchitis cases, 1940 to 1949. In a group of 1,077 patients, tracheotomies were performed in 86 (8 per cent) with a mortality of 11.6 per cent. Pneumothorax resulted in 10.4 per cent of the tracheotomized cases; there were two bilateral cases, both fatal, and 7 unilateral cases with one death. In the unilateral cases, 6 out of 7 occurred on the left side. This latter coincides with the reports of other observers. Simpson[8] compares the former average of about 50 per cent mortality with more favorable results in recent years, incident to the use of the new drugs and earlier tracheotomy, but he notes that in a group of 64 cases requiring tracheotomy, 12 were fatal (18 per cent). Bronchial obstruction was the most frequent cause of death.

SUMMARY AND CONCLUSIONS

1. Acute laryngotracheobronchitis is a fulminating medical and surgical emergency because of the danger of obstruction to breathing. The rather sudden onset, high fever, stridor, and extreme toxicity distinguish the infection from croup. Etiological factors are discussed; the condition can be rapidly fatal; serious signs and symptoms are given.

2. Early recognition, hospitalization, and proper medical treatment decrease the need for surgical in-

tervention. Cool humidity by various means is discussed and is favored. Oxygen concentrations of 40 to 50 per cent are necessary. The use of antibiotics is outlined with reasons for the choice of penicillin, streptomycin, and aureomycin; other medical measures are mentioned.

3. The surgical treatment of acute infective laryngotracheobronchitis becomes a question of good judgment and weighing the factors: (1) the degree of airway obstruction, and (2) the ability of the circulatory system to stand the overload. Tracheotomy is the procedure of choice, and it should be performed relatively early rather than as a last resort. The indications for operation, technique, aftercare, and complications have been described and the results of treatment have been discussed.

BIBLIOGRAPHY

1. Jackson, C., and Jackson, C. L.: Diseases of the nose, throat and ear, W. B. Saunders Company, 1946.
2. Gilbert, J. G.; Kosnetz, J.; Rosenthal, I. M., and Mazzarella, L.: Infectious nondiphtheritic croup, Arch. Otolaryng. 55:566-581 (May) 1952.
3. Gorman, J. R.: Use of a wetting agent in laryngotracheobronchitis, Laryngoscope 60:510-514 (May) 1950.
4. Hollinger, P. H., and Johnston, K. C.: Factors responsible for laryngeal obstruction in infants, J.A.M.A. 143:1229-1232 (Aug. 5) 1950.
5. Morgan, E. A., and Wishart, D. E. S.: Laryngotracheobronchitis: statistical review of 549 cases, Canad. M.A.J. 56:8-15 (January) 1947.
6. Crowe, S. J.: Editorial comment to reference 5, Year Book of Eye, Ear, Nose and Throat, 1947, pp 440-441.
7. Priest, R. E.: Treatment of laryngotracheobronchitis, Postgrad. Med. 8:489-496 (December) 1950.
8. Simpson, J. R.: Management of laryngotracheobronchitis, Arch. Otolaryng. 50:724-731, (December) 1949. 8a. Ibid. Discussion by Hollinger, P. H.
9. Everett, A. R.: Acute laryngotracheobronchitis: an analysis of 1175 cases with 98 tracheotomies, Laryngoscope 61:113-123 (February) 1951.
10. Galloway, T. C.: Postural treatment of acute laryngotracheobronchitis, J. Mich. M. Soc. 36:969 (Dec.) 1937.

The Diagnosis of Lead Poisoning

J. L. Morgan, M.D.
Emporia, Kansas

INTRODUCTION

Lead was one of the first metals used by man. Lead intoxication or lead poisoning, the terms being synonymous, was known to the Greek, Roman, and Arabian physicians long before the time of Christ.[9]

Today, lead poisoning is rarely seen outside of the "lead industries." The non-occupational exposures are the ones we all heard of as medical students, being presented to us as someone's diagnostic accomplishment. Typical of these case reports was the comatose child who was found to have eaten leaded paint from his crib and the woman with colic who had burned discarded storage batteries. Some of these sporadic poisonings are listed in Table 1.

TABLE 1

CAUSES OF NON-INDUSTRIAL LEAD POISONING

1. Burning storage batteries.
2. Infants chewing paint from cribs.
3. Lead in rubber nipples and toys.
4. Amateur painting at home.
5. Snuff wrapped in lead foil.
6. Wines and beer made at home.
7. Galvanized nails held in the mouth.
8. Drinking of "canned heat."
9. Handling of lead seals.
10. White lead used as a fingerprinting powder.

These poisonings are rare now, thanks to careful public health measures. In this regard, it is probably news to most of us that lead poisoning is a reportable disease in Kansas.

By far the greatest lead poisoning problem today is encountered in industries. Lead poisoning is second only to dermatoses in the frequency with which it causes disability of workers. Several hundred occupations have been called the "lead industries." A few of these are listed in Table 2.

TABLE 2

WORKERS EXPOSED TO LEAD

1. Storage battery assemblers	11. Ink makers
2. Auto "stream liners"	12. Rubber workers
3. Painters	13. Insecticide manufacturers
4. Welders	14. Tetraethyl lead workers
5. Lead smelters	15. Plumbers
6. Pottery makers	16. Blacksmiths
7. Enamelers	17. Air craft workers
8. Printers	18. Gardeners
9. Solderers	19. Tree sprayers
10. Brass workers	20. Canners

Of these, the storage battery plants account for the greatest number of lead poisoning casualties. In all of these industries, the workers are known to be exposed to lead and are assumed to have absorbed a certain amount of lead. The diagnosis in these workers depends on a differentiation between lead absorption, which is nondisabling, and lead poisoning, which is disabling.

PATHOGENESIS

Some confusion exists in the use of the terms acute

Medical Examiner, Globe Union, Inc. (battery company).
Paper presented at the Regional Meeting, American College of Physicians, Kansas City, Kansas, March 20, 1953.

and chronic lead poisoning. I believe with others[5] that so-called "chronic" lead poisoning is a misnomer and such previously labeled states represent in fact a series of bouts of acute poisoning. Cantarow and Trumper[8] in 1944 exhaustively presented the studies leading to our present concept of the pathogenesis of lead poisoning.

Lead products may be inhaled, swallowed, or absorbed through the skin. By far the most important means of entry is the respiratory tract. Most lead compounds can be demonstrated in the tissues of experimental animals a few hours after intratracheal instillation. Finer particles of lead are assimilated most rapidly, supposedly by phagocytosis; however, volatile lead products, particularly tetraethyl lead, may be absorbed directly from the skin or lungs. Swallowed lead is many times less toxic than inhaled lead, 84 per cent of swallowed lead being excreted in the feces.

Regardless of the portal of entry, lead is distributed throughout the viscera immediately after entering the blood stream. The liver, muscles, and spleen are the usual sites of early deposition, an exception being tetraethyl lead which is readily soluble in fats and concentrates in the central nervous system. A few days after absorption, lead is found in ever-increasing quantities in the bones where it is stored as the phosphate, regardless of the compound which was originally absorbed. There is a parallelism between the behavior of calcium and of lead. The factors which favor the deposition of calcium also favor the deposition of lead, and the same applies to mobilization. Stored lead in the bones is not disabling, but it may be released during subsequent illnesses and then cause symptoms. The human body can accommodate large quantities of lead, when gradually absorbed, without producing symptoms. This state of being "in equilibrium" with lead is spoken of as lead absorption and is clinically important only as it may be a forerunner of lead poisoning. When absorbed lead causes symptoms, it is assumed that organ damage has occurred. Lead poisoning most significantly affects the hematopoietic system, the muscles, and the nervous system (Table 3).

TABLE 3

PATHOGENESIS OF LEAD POISONING

Hematopoietic System
RBC—hemolysis, stippling
Hgb—faulty synthesis
WBC—lymphocyte shift
Smooth Muscle—direct effect
Voluntary Muscle—phosphocreatine metab.
Central Nervous System—
Brain—ganglion cells, capillary damage, exudation, hypertensive damage (?)
Meninges—irritation
Spinal Cord—ant. horn cells degen.
Other Systems Involved—vascular tree (?), kidneys (?), joints (?), gums

I. *Hematopoietic System:*

a. Red blood cells:

Lead causes an increased hemolysis of the red blood cells, and in consequence anemia occurs.

Increased basophilic stippling of the red cells occurs. This is a nonspecific reaction of the red cells which may follow a number of stimuli as listed in Table 4.

TABLE 4

CAUSES OF "STIPPLING"

Infectious disease—e.g. pneumonia
Blood diseases—anemia, leukemia, Hodgkin's disease
Advanced malignancy
Poisoning—lead, mercury, arsenic, aniline, nitrobenzol, carbon disulfide, benzol, gasoline, phenyl-hydrazine
Cirrhosis of the liver

Statistically, however, stippling is most often due to lead. The granules in the cells are not precipitated lead, but are of cytoplasmic origin. Usually stippled cells disappear from the blood two to four weeks after cessation of exposure to lead.

b. Hemoglobin:

Lead interferes with the synthesis of hemoglobin,[17] lowering the hemoglobin level and causing an organic pigment normally contained in the heme component of hemoglobin to be excreted as coproporphyrin III in large amounts in the urine. Most reports, and my experiences, indicate that the urinary porphyrin test is usually an earlier guide to lead damage than is the stipple count.[7, 11, 17] Just as increased stippling is not specific for lead poisoning, neither is an increase in the urinary porphyrin level. Some of the conditions noted to cause this finding are listed in Table 5.

TABLE 5

CAUSES OF PORPHYRINURIA

Infectious dis.—pneumonia, poliomyelitis, syphilis
Liver dis.—hepatitis, cirrhosis
Blood dis.—aplastic anemia, hemolytic anemias, Hodgkin's dis., Addisonian anemia, polycythemia
Deficiency dis.—pellagra, sprue
Skin dis.—eczema, lupus
Occupational exposure—lead, mercury, arsenic, beryllium, selenium, phosphorus, aniline, carbon tetrachloride, benzene
Drugs—alcohol, salicylates, phenacetin, barbiturates

An increase of urinary porphyrins due to stimuli other than lead poisoning is small. Normal urine, for instance, contains 10 to 30 micrograms of porphyrin per liter; in benzene poisoning this level may be 255, while in lead poisoning it is over 3,500 micrograms per liter.[17]

c. White blood cells:

The total white cell count in lead poisoning is

usually normal. Lead damage has been felt to alter the ratio of large mononuclear cells to small lymphocytes.[15]

II. *Smooth Muscle:*

The direct effect of lead on the smooth muscle of the intestinal tract is thought to explain the constriction of the small intestine and the resultant colicy pain which is most characteristic of all the symptoms of lead poisoning. X-ray studies during colic do show hypermotility and spasm of the small intestine with subsequent atonicity as the pain eases.

III. *Voluntary Muscle:*

"Lead palsy" or the muscle weakness seen in lead poisoning is due to a direct effect of lead on the voluntary muscles. Specifically, lead interferes with the resynthesis of phosphocreatine. Because this chemical effect hinders the recuperation of muscles following use, the most exercised muscles, such as the wrist, are involved.

IV. *Central Nervous System:*

a. Brain:

The ganglion cells in the brains of cats are damaged when the cats have been experimentally poisoned with lead. Other studies have indicated that cerebral symptoms may be dependent on capillary damage with subsequent exudation. The cerebral manifestations may mimic hypertensive encephalopathy clinically, and the frequent occurrence of hypertension with this presenting picture has been thought to indicate hypertension, secondary to lead, as the causative factor.

Certainly any of these mechanisms could account for the convulsions and coma which typify cerebral damage in humans.

b. Meninges:

There is irritation of the meninges, as evidenced by increased cerebrospinal fluid pressure, increased protein, and pleocytosis.

c. Spinal cord:

Degeneration of the anterior horn cells has been noted at autopsy, and this may explain the muscle atrophy occasionally seen in lead poisoning.

V. *Other Systems Involved:*

Less frequent and more controversial are the effects of lead on the vascular tree, particularly its association with hypertension.[1] There is no doubt that hypertension occurs frequently in association with lead colic, but this may be purely a reaction to pain. Too, as mentioned before, its more than casual frequency in connection with cerebral symptoms has been thought to indicate that lead causes hypertension and the cerebral symptoms follow. In studies of patients exposed to lead for many years, there is no convincing evidence that hypertension is more frequent than would be expected in an unexposed group.

Damage to the renal tubules may occur in massive lead poisoning, the changes being similar to those produced by other heavy metals. Repeated episodes of poisoning, over a long period, have been thought by some to cause nephrosclerosis as a component of generalized vascular damage. Oliguria, hematuria, and albuminuria may be noted coincident with the appearance of symptoms of lead poisoning, these urinary findings usually clearing as the symptoms are relieved.

Joint pains do occur during lead poisoning, and no morphological changes in the joints have been found to explain this. Stored lead in the bones does not cause these or other symptoms.

Lead combines with decomposed food and gum tissues to form the typical "lead line" in the gums. This consists of a number of minute granules of lead sulfide in the gums. Poor dental hygiene is necessary for its formation, and even with severe lead poisoning the "lead line" may be minimal if the gums are scrupulously clean.[2, 3]

CLINICAL MANIFESTATIONS

TABLE 6

CLINICAL MANIFESTATIONS
History
Symptoms:
Abdominal colic
Muscle weakness
Joint pains, diarrhea, nausea, fatigue, metallic taste
Central nervous system symptoms
Physical signs:
Rigid abdominal wall with colic
Elevated blood pressure with colic
Muscle weakness
Pallor, lead line
Central nervous system signs

I. *History:*

In practice, a history of exposure to lead is often the clue to the diagnosis.

II. *Symptoms:*

By far the most common symptom of lead poisoning is abdominal colic.[4, 5] This may be sudden in onset, but it is often preceded by vague abdominal pains and constipation. The pain is severe and griping and is located in the mid or lower abdomen. Usually each colic lasts several minutes, and the colics recur several times hourly. A sense of pressure may persist between colics. Vomiting is not a prominent feature, though retching is common. The colic is promptly relieved by calcium salts given intravenously.

Muscle weakness, particularly wrist weakness, may

accompany colic, though more often it is a later symptom.

It is interesting that Benjamin Franklin[10] wrote a letter in 1786 which graphically describes these two most important symptoms. He wrote that when he was a boy he heard of a complaint from North Carolina against New England rum. It "poisoned their people, giving them the dry belly-ache, with a loss of the use of their limbs. The distilleries being examined on the occasion, it was found that several of them used leaden stillheads, and the physicians were of the opinion that the mischief was occasioned by that use of lead."

Less frequent and less characteristic symptoms are joint pains, diarrhea, nausea, fatigue, and a metallic taste.

An exception to the usual symptom picture is a predominant, early involvement of the central nervous system. This is now rarely seen, but it does occur in infants, regardless of the lead product involved, in adults poisoned suddenly with massive amounts of any lead product, and in adults poisoned by tetraethyl lead. In each of these instances, the symptoms may be sudden in onset with a rapid progression to convulsions, coma, and death. Insomnia, restlessness, headache, and depression may occasionally constitute a prologue to such central nervous system involvement.

III. *Physical Signs:*

There are very few specific signs of lead poisoning. During bouts of colic, the abdominal muscles are rigid, and the blood pressure is often elevated. The patient evidences distress, often doubling up and pressing his abdomen, which procedure seems to give a measure of relief. Between bouts of colic, the abdomen is doughy and not tender to moderate palpation.

Muscle weakness, particularly of the extensor muscles of the wrists, may be demonstrated by having the patient attempt dorsal extension against resistance. There are no accompanying sensory changes.

Signs of lead absorption, which may be present in the absence of symptoms, but which are almost invariably present during lead poisoning, are pallor and a lead line in the gums. The lead line is seen with a hand lens as a series of minute blue-black particles on the margin of the gums, and not on the teeth.

In the rare instances of central nervous system involvement, the signs may mimic encephalitis in their variability. Convulsions, delirium, blindness, aphasias, and psychoses may come on very rapidly and proceed through coma to death.

LABORATORY FINDINGS

A summary of the laboratory tests altered in lead exposure, absorption, and poisoning is listed in Table 7. An asterisk marks those tests which are most important to the practicing physician.

TABLE 7

ABNORMAL LEAD VALUES

Exposure:	
Air	more than 1.5 mg/10 cu. liters
Absorption:	
Red Cell count*	less than 4.5 mil/cu. mm.
Hemoglobin*	less than 80%
"Stipple" count*	more than 5/50 HPOIF**
Blood lead content	more than .08 mg/100 cc.
Urine lead content	more than .08 mg/liter
Bone x-rays in children	diaphyseal deposition
Damage (?):	
"Stipple" count*	more than 20/50 HPOIF**
Urine porphyrin test*	pink fluorescence

* Easily performed in office laboratory.
** HPOIF—High power oil immersion fields.

I. *Measure of Exposure:*

The measure of lead particles in the air is the best laboratory reflection of the degree of exposure to lead. Actually, this test is valuable in determining the lead hazard in particular industries, but it is outside the means of the practicing physician. It is interesting that less than 1.5 mg/10 cu. liters is not considered an abnormal concentration, implying that we are all exposed to lead "normally," albeit small amounts.

II. *Measures of Absorption:*

The tests altered by lead absorption are the red cell count, the hemoglobin determination, the stipple count, the quantitative blood, the quantitative urine lead tests, and (in children) x-rays of the bones.

a. Red blood cell and hemoglobin determination:

The anemia is hypochromic, though seldom severe. The red cell count is usually less than four and one-half million but rarely less than four million, and the hemoglobin is usually less than 80 per cent, though rarely less than 60 per cent.

b. Stippling of the red blood cells:

Stippling of the red cells is best seen by examining a single layer of cells stained with Wright's stain. They may be tabulated in terms of stippled cells per 50 high power oil immersion fields (abbreviated HPOIF) or as stippled cells per million red cells. Over five stippled cells per 50 fields is evidence of absorption.

c. Quantitative blood lead level:

The concentration of lead in whole blood bears no consistent relationship to the appearance of clinical manifestations.[1, 6] Symptoms may be present at low concentrations or absent at high concentrations.[16] This discrepancy has been suggested to be due to the relative distribution of lead between plasma and red cells[8] and the inability of the commonly used tests to differentiate these fractions. Probably less than

10 per cent of the blood lead is in the plasma, and this fraction is in turn divided into a larger non-dialyzable colloid form and a smaller dialyzable form which is the portion of importance in causing lead damage. Since these divisions of blood lead represent such minute amounts, even in the aggregate, the test aims to determine only the whole blood content.

d. Quantitative urine lead level:

As in the instance of blood lead level, an elevated urine lead level is indicative of increased absorption, transportation, and excretion, and is not diagnostic of lead poisoning.[1, 11, 13] In most cases of frank lead poisoning, the urine lead value is increased, but this may be less than that seen in many instances of simple absorption. The urine content does not bear a constant relation to the blood level, renal function being reflected in the urine determination. The urine test, like the blood test, measures minute quantities of lead, so the contamination by the patient in voiding and collecting the sample is a major factor. The state of hydration of the patient is reflected when the figures are recorded in milligrams per liter.

In both the blood and urine determinations, special lead free glassware must be used, and I feel that these tests are not suited to an office laboratory. The Kansas State Board of Health will make a study of an industrial plant, if requested, and urine and blood determinations may be a part of that study, but the state laboratories do not accept individual samples of blood or urine sent in for lead determinations.[14]

e. X-rays:

X-rays of the growing ends of the bones may show a lead deposit in children who have absorbed much lead, though in adults x-rays are of little help.

III. *Measure of Damage:*

Though the tests for absorption are usually positive in lead poisoning, the tests more specifically indicating damage are excessive stippling of the red cells and a positive urinary porphyrin test.

a. Stipple count:

The stipple count is probably a better guide to lead damage than the blood or urine lead level. Stippling may be absent in the rare cases of sudden massive ingestions of lead with a rapid onset of symptoms, but usually over 20 stipple cells per 50 oil immersion fields are present in lead poisoning, and serial determinations show an increase of stippled cells as poisoning becomes manifest.

b. Semi-quantitative urine porphyrin determination:

The urinary porphyrin test is a simple determination applicable to the average practitioner's laboratory. I use the method of McCord,[12] slightly modified, as illustrated in Table 8.

TABLE 8

URINARY PORPHYRIN DETERMINATION

1. Pipette 10 cc. of a "random" sample of urine into a screw top tube.
2. Add 2 drops of glacial acetic acid.
   Add 2 drops of hydrogen peroxide.
   Add 2 *cc.* of ether.
3. Mix gently in screw top tube.
4. Let stand 30 minutes.
5. View in a dark room using an ultra-violet lamp.
6. Record any pink fluorescence of the surface ring as POSITIVE.

There is no correlation between the quantitative amount of lead in the urine and the presence of a positive porphyrin reaction.[12] The test may vary from positive to negative in the course of a few months time, or it may stay positive long after cessation of exposure to lead when damage supposedly persists.[6, 9]

SUMMARY

Lead poisoning occurs when absorbed lead produces sufficient damage to cause disabling symptoms.

The diagnosis of lead poisoning is based on the history, symptoms, and signs, coupled with the laboratory findings, and is not based on any one specific laboratory test.

TABLE 9

DIAGNOSIS OF LEAD POISONING

History of exposure
- Usual—occupational
- Rare—non-occupational

Symptoms
- Usual—abdominal colic
- Occasional—wrist weakness
  joint aches
- Rare—cerebral symptoms

Signs
- Lead line
- Pallor

Laboratory tests
- Hemoglobin
- Stipple count
- Urinary porphyrin test

Certain simple tests are of practical value in confirming a clinical diagnosis of lead poisoning. These tests are the hemoglobin determination, the stipple count, and the urinary porphyrin test.

The urinary porphyrin test is much easier to perform than the stipple count. The porphyrin test has a much clearer "end point," and it is usually positive before the stipple count becomes abnormally elevated. The quantitative advantages of the stipple count, however, would seem to preclude its complete replacement by the porphyrin test.

BIBLIOGRAPHY

1. Anonymous: Occupational lead exposure and lead poisoning, New York, American Public Health Association, 1943, pp. 67.
2. Belknap, E. L.: Control of lead poisoning in the worker, J.A.M.A. 104:205-210, 1935.
3. Belknap, E. L.: Lead poisoning, presented at the 25th annual meeting of the American Association of Industrial Physicians and Surgeons, 1940.
4. Belknap, E. L.: Differential diagnosis of lead poisoning, J.A.M.A. 139:818-823, 1949.
5. Belknap, E. L.: Personal communications, 1949, 1950.

6. Belknap, E. L.: The diagnosis and treatment of lead poisoning—some new concepts, presented at the 24th annual meeting, Lead Industries Association (April 18-19) 1952.
7. Brooks, A. L.: An appraisal of a urinary porphyrin test in the detection of lead absorption, Indust. Med. and Surg. 20: 390-92 (Sept.) 1951.
8. Cantarow, Abraham, and Trumper, Max.: Lead poisoning, Baltimore, Williams and Wilkins Co., 1944, pp. 264.
9. Fullerton, M. B.: Value of hematology in the diagnosis of chronic plumbism, Brit. Med. J.:117-19 (July 19) 1952.
10. Franklin, Benjamin: Letter dated July 31, 1786. In: van Doren, Carl: Benjamin Franklin's autobiographical writings, New York, Viking Press, 1945, pp. 671-673.
11. Harrold, Gordon C.; Meek, Stuart F., and Padden, David A.: A coproporphyrin III test as a measure of lead damage, Arch. Indust. Hygiene and Occup. Med. 6:24-31 (July) 1952.
12. McCord, Carey P.: The porphyrins: the significance of porphyrins in occupational disease, Indust. Med. and Surg. 20: 185-190 (April) 1951.
13. Maloof, C. C.: Role of porphyrins in occupational diseases, Arch. Indust. Hygiene and Occup. Med. 1:296-307, 1950.
14. Mayes, J. L.: (Director, Industrial Hygiene Section, Kansas State Board of Health), Personal communications, 1952.
15. Shiels. D. O.: Influence of lead absorption on the ratio of large to small lymphoid cells, Med. J. Australia 2:205-211 (Aug. 5) 1950.
16. Smith, J. Earl; Lewis, B. W., and Wilson, Herbert S.: Lead poisoning: a case finding program, Am. J. Public Health 42:417-421 (April) 1952.
17. Waldman, R. K., and Seideman, R. M.: Reliability of the urinary porphyrin test for lead absorption. Arch. Indust. Hygiene and Occupational Med. 1:290-295, 1950.

The Effect of Altitude on the Pathogenesis Of Tuberculosis

William F. Casteen

Sophomore Student, University of Kansas School of Medicine

This paper seeks to make inquiry, by means of a survey of the literature, into the rationale of high altitude in the treatment of tuberculosis, by analysis of the possible effects of altitude on the pathogenesis of tuberculosis. Information will be drawn from three general fields: cellular physiology and biochemistry, animal experimentation, and clinical and laboratory observation and correlation.

The earlier proponents of altitude therapy based their opinions not upon any known effect of altitude on the disease process, or upon any scientific rationale, but rather upon clinical observation. A sampling of literature containing reports of these observations indicates that most such reports covered at most a few cases, and they are seemingly cases with which the author was particularly impressed. Large scale surveys involving large numbers of patients with comparable disease, under comparable conditions, were not made. Many of the proponents of altitude in the treatment of tuberculosis believed that benefit resulted not from altitude directly, but from increase in intensity of the sun's rays, or other forms of therapy with which they were concerned.[1, 2, 3, 4]

In more recent times climatic therapy has largely fallen into disrepute. Articles in medical literature pertaining to these forms of therapy, and more particularly to altitude therapy, are indeed few in number. Interest and effort have been turned to chemotherapy and other methods based upon a more sound rationale.

Lately, however, evidence has come to light which provides a sound rationale for altitude in the treatment of tuberculosis. At least one modern compilation of data on a relatively large and complete scale has indicated that altitude has a definite beneficial effect on tuberculosis.[12]

If any factor is to impede or halt the progress of tuberculosis in the body, it must do so by one of two means, by inhibiting the growth or metabolism of the tubercle bacillus, or by increasing the ability of the cellular defense mechanisms of the body to contain and kill the invading organism.

It has long been known that the tubercle bacillus is sensitive to the oxygen tension of its environment.[5, 6, 7, 9] In 1938 the experiments of Walter Kempner clearly outlined this relationship and put it on a quantitative basis.[6] Kempner's experiments may be graphed to show a remarkably smooth curve indicating the rate of metabolism of tubercle bacilli at varying oxygen tensions. The curve shows that at a certain point reduction in oxygen tension produces a precipitous drop in the rate of metabolism of the bacilli.

At sea level the oxygen tension in the alveolar spaces is about 100 mm. Hg., and Kempner has estimated, at sea level, the oxygen tension inside a tubercle to be approximately 50 mm. Hg. This reduction in oxygen tension is due to the fact that a gradient of tension must exist to achieve transfer of oxygen, since a gas will diffuse only from a region of higher tension to one of lower tension. A series of tension gradients exists between alveolar spaces and the inner layers of a tubercle. If the oxygen tension in the alveolar spaces is reduced, the series

Research paper from the Department of Pathology.

of tension gradients must be maintained, and the decreased oxygen tension transmitted to the end of the line, the inner layers of the tubercle. The body reacts to decreased oxygen tension in the atmospheric air by an increase in the number of circulating red cells and an increase in the concentration of hemoglobin. This permits the circulatory system to maintain the quantity of oxygen transported in the face of the reduced tension or pressure.

The cellular reaction to the tubercle bacillus and the formation of the tubercle are well known. The mononuclear cells which constitute the most prominent part of the body's reaction to the invasion of tubercle bacilli, and its principal means of defense, seem to encounter great difficulty in the phagocytosis of tubercle bacilli and also their destruction after phagocytosis. A mononuclear cell may phagocytose a tubercle bacillus and migrate from the focus of infection only to be overcome by its ingested bacillus and thereby set up a new focus of infection.[8] The mononuclear cells form themselves into a wall about the bacilli.

One might offer many explanations as to how this capsule of mononuclear cells tends to contain the infection and work to the detriment of the bacilli. One explanation might be that this process intends the deprivation of the bacilli of oxygen. If the mononuclear cells cannot readily destroy the bacillus by phagocytosis, this might well be their principal means of combating the tubercle bacillus. The mononuclear cells form themselves many layers thick into the wall of the tubercle. Each cell removes oxygen from its environment so that in successive regions toward the center of the tubercle the oxygen tension must become progressively reduced. Indeed, oxygen at the innermost points must diffuse through the protoplasm of many successive layers of mononuclear cells. This effect is enhanced by the fact that the tubercle is an avascular lesion, in contradistinction to the lesions of most infectious processes.

The zone of lymphocytes which accumulates around the periphery of the epitheloid cells, although taking no part in the phagocytosis of the bacilli, must certainly aid in reducing the oxygen tension on the inside of the tubercle, since they will reduce the amount of oxygen diffusing into the tubercle by metabolizing a part of the oxygen available.

From an evolutional point of view, the oxygen relationship might be considered as a reason for the avascularity of the tubercle. The benefit derived from this avascularity is difficult to explain on any other grounds. By the reduction of oxygen tension alone, however, the tubercle bacilli cannot be destroyed, because it has been shown by in vitro studies that so long as the slightest traces of oxygen are present the tubercle bacilli will remain viable.[7]

If an animal infected with tuberculosis was to ascend to an altitude of 10,000 feet where the oxygen tension of the atmosphere is 110 mm. Hg., and of alveolar air is 62 mm. Hg., a conservative estimate of the oxygen tension within a tubercle would be in the neighborhood of 30 mm. Hg. Interpreted in terms of the results of Kempner's in vitro experiments, this indicates that the metabolic rate of the bacilli would be approximately 65 per cent of the rate at sea level. It is in this range of oxygen tension that the more sharply dropping portion of Kempner's curve is encountered.

Since the general public ascends to the summit of Pike's Peak at about 14,000 feet, even altitudes higher than 10,000 feet (or synthetic altitudes) would seem plausible in therapy with benefit increasing at a steeply rising rate. Altitudes of 6,000 feet have rarely been employed in the treatment of tuberculosis in the past. The reduction of oxygen tension afforded by this and lower altitudes is not sufficient to reach the more sharply dropping portion of Kempner's curve, and hence the reduction in the metabolic rate of the tubercle bacillus at such an altitude is quite small. This may be an explanation of the controversial results of such treatment.

Animal experiments give conflicting evidence. Barach's[9] experiments with rabbits were inconclusive. David,[10] experimenting with rabbits and dogs, found that low oxygen tension had a definite beneficial effect.

It is well known that composition of the white cell components of blood changes with altitude. The percentage of lymphocytes increases, and the percentage of neutrophiles decreases with an increase in altitude. Although there has been a considerable difference of opinion as to whether differential blood counts and total white cell numbers are of value in indicating anything in tuberculosis, a number of investigators, having done extensive work on the question, believe that there is a correlation between a high neutrophile count and failure of the body in containing and recovering from the disease.[11, 12] Many possible sources of error have been scrutinized and evaluated in arriving at this conclusion.

Boissevain and Chapman[12] reported in 1941 the results of an extensive study of blood counts at two sanatoria; one, Mt. McGregor in New York, at an elevation of 1,000 feet, and the other, Cragmoor in Colorado Springs, at an elevation of 6,000 feet. The care in both sanatoria was comparable. These investigators reasoned that if a given patient was moved to a higher altitude, the number of neutrophiles in his blood count would decrease, and if altitude was not an influencing factor his prognosis should remain the same. If the neutrophile count is used as a measure of prognosis, the decrease would

give a false indication of better prognosis. In other words, the prognosis for a given neutrophile count should be better at Mt. McGregor than at Cragmoor.

Extensive blood counting had been done on approximately 200 patients at each sanatorium. The results found by these investigators was opposite to that deduced above. The tabulated results of their investigations may be plotted as a graph which presents strikingly consistent and smooth curves. With a neutrophile count of 7,000-8,000, 71 per cent of the patients at Mt. McGregor subsequently died, whereas only 18 per cent subsequently died at Cragmoor. These investigators present evidence to rule out humidity, sunshine, and other factors as the critical agent in producing the difference in prognosis, leaving altitude as the sole remaining agent. A rigorous statistical analysis was used in interpreting the data.

SUMMARY

The tubercle bacillus is sensitive to a decrease in the oxygen tension of its environment, and it is increasingly inhibited as the oxygen tension is reduced. As long as the slightest trace of oxygen remains, this factor will not effect the death of the bacillus. It is possible that the basic and most important defense process of the body against the bacillus, the formation of the tubercle, is an attempt by the body to reduce the oxygen tension available to the bacillus and thereby inhibit its metabolism and reproduction.

Whether or not the reduction of oxygen tension available to the tubercle bacillus is the most important result of tubercle formation, it must produce some degree of inhibition of the bacillus. Whether or not this aspect of the body's defense mechanism against the tubercle bacillus can be appreciably reinforced by decreasing the oxygen tension in the environment of the entire body has neither been proved nor disproved. Animal experiments have given conflicting and inconclusive evidence.

Clinical observation and correlation by laboratory studies of the highest quality at two large sanatoria have indicated that altitude has a definite beneficial effect in the treatment of tuberculosis. Further animal experimentation and clinical evaluation are needed.

BIBLIOGRAPHY

1. Bernhard, O.: Strahlentherapie 9:520 (Sept. 10) 1919 (abstracted).
2. Rollier, A.: Am. Rev. Tuberc. 8:489. (Feb.) 1924.
3. Hill, L.: Brit. J. Tuberc. 17:155 (October) 1923 (abstracted).
4. Bacmeister, A.: Ztschr. f. Tuberk. 37:161 (Dec.) 1922 (abstracted).
5. Dubos, Rene: J. of Exp. Med. 97:357. (March) 1953.
6. Kempner, Walter: Am. Rev. Tuberc. 40:157. (Aug.) 1939.
7. Novy, F. G., and Soule, M. H.: J. Infect. Dis. 36:168, 1925.
8. Permar, Howard H.: Am. Rev. Tuberc. 9:507. (Aug.) 1924.
9. Barach, Alvan C.: Am. Rev. Tuberc. 9:534. (Apr.) 1926.
10. David, O.: Ztschr. f. exper. P. u. Therap. 11:239. 1912 (abstracted).
11. Medlar, E. M., and Kastlin, G. J.: Am. Rev. Tuberc. 16:706. (Dec.) 1927.
12. Boissevain, C. H., and Chapman, E. N.: Am. Rev. Tuberc. 44:58. (July) 1941.

The basic characteristic of the Welfare State is that it cuts the historic, traditional tie between a man's income and his own effort.

For some services, we all recognize that it is desirable to cut this tie—public libraries, free public education and certain public health services. But there is no substitute for individual responsibility and incentive and the discipline of the market in the overwhelming majority of human and economic relationships.

An English observer put it succinctly this way: "The central objection of the Left to the reduction of food subsidies is that it hits the poor more than the rich. But the purchase of anything absorbs more of the income of the poor man than of the rich. This is the main reason why men strive to avoid poverty. They can avoid poverty only by working harder and producing more; and their efforts tend to make goods cheaper for everybody."

—Emerson P. Schmidt, Director, Economic Research
United States Chamber of Commerce

Sixth Annual Mid-West Cancer Conference

Broadview Hotel, Wichita, Kansas April 1 and 2, 1954

GUEST SPEAKERS

A. N. Arneson, M.D.
St. Louis, Missouri

Louis H. Clerf, M.D.
Philadelphia, Pennsylvania

Warren H. Cole, M.D.
Chicago, Illinois

Hugh F. Hare, M.D.
Los Angeles, California

N. L. Higinbotham, M.D.
New York, New York

Richard H. Overhold, M.D.
Brookline, Massachusetts

Cornelius P. Rhoads, M.D.
New York, New York

Arthur P. Stout, M.D.
New York, New York



Warren H. Cole, M.D.

Head, Department of Surgery, Illinois College of Medicine; Graduate, Washington University School of Medicine, 1920; Diplomate, American Board of Surgery; Fellow, American College of Surgeons.

Professor of Pathology, Columbia University College of Physicians and Surgeons, New York; Graduate, Columbia University, 1912; Diplomate, American Board of Pathology.



Arthur P. Stout, M.D.

Biographical sketches and pictures of additional speakers will be featured in subsequent issues of the Journal.

Sponsored by
American Cancer Society, Kansas Division
Committee on Control of Cancer, Kansas Medical Society

PRESIDENT'S PAGE

DEAR DOCTOR:

By the time this issue of the JOURNAL reaches you, it will be 1954. For many of you it will mean one more year toward a lifelong ambition of a successful career in the practice of medicine. For many of us it will mean one less year to complete the things that 20 or more years ago we knew in our own minds would be completed, all wrapped up in a neat package, tied with a blue ribbon, and put upon a shelf of our memory as a prize for a job well done. And just the fact that the package isn't finished and wrapped up for many of us, even after 20 or 30 years of practice, is a good reason for all of us to stop and ponder for a few moments.

In the lay press and in our conversations with lay people, we are constantly being reminded of the "family doctor." I have, among my personal friends, many who say that they wouldn't know who to call if they suddenly became ill. They bewail the passing of the family doctor in one breath, but they are the same people who, a few years ago, would go out of town to see a diagnostician, or who now go to some distant place to go through a clinic. They may have a diagnosis made and then come home to a friend for an operation, for he is a specialist in surgery and they know him and have confidence in him as a friend and in his ability as a surgeon.

I am sure that the public as a whole is totally appreciative of the age of specialization in medicine and the wonderful advances that have been made in the diagnosis and treatment of disease in the past half century. I am equally sure that not one of the people who criticize the medical profession would be willing to go back to the "family doctor" of 50 years ago, if he had no more to offer them now than he had then. But they would like the companionship and the friendly banter that have been lost to them in the changing of the times and the necessary speeding up of all of our activities.

The old family doctor was a past master in public relations. That is how he got his patients in the first place, and that is how he kept them. In the increasing demands upon the medical profession in the past 10 years, a lot of the niceties of the old "family doctor" have been lost in the shuffle. It is that particular fact that has cast more aspersions on the medical profession than any other one thing. It is a little thing, but it would be easy to correct. It would not take very much of our time. In fact, it would probably lessen the time that we have to spend with patients because they would again develop that abiding faith in us that they had in the "family doctor" of yesteryear.

We hear a lot about public relations. Public relations, as far as the medical profession is concerned, begins in your own office, with you, with your office personnel, and in your social contacts. When this art has again been fully mastered, we will have little need for departments of public relations in our local, state, and national offices.

I wish for each and every one of you and your families, health and happiness, and for you a successful year in the practice of medicine.



EDITORIAL COMMENT

Polio Vaccination Field Trials

On Friday, December 18, in the offices of the Shawnee County Medical Society, a meeting was held that might one day prove to be of historical significance in medicine's attempt to control the incidence of poliomyelitis. On that date, some 30 physicians from various parts of Kansas, representing the Committee on Child Welfare of the Kansas Medical Society, the specialties of internal medicine and orthopedics, as well as pediatrics, officers of at least four among the larger county medical societies of this state, and public health officials, assembled to hear Carl Potthoff, M.D., regional medical director of the National Foundation for Infantile Paralysis.

He suggested that one and possibly two Kansas counties might be included in the national field trial immunizing program to be conducted in 1954, beginning in January. At the conclusion of this meeting, the Committee on Child Welfare met and endorsed the program as explained below. Subsequent endorsement was given by the Council of the Kansas Medical Society. Therefore, if approved by the National Foundation, Kansas will participate in this venture.

Dr. Potthoff spent much time explaining the processes involved in the immunization program and the materials to be used. Jonas Salk, M.D., of the University of Pittsburgh, has developed what appears to be an immunizing agent, and it is his product that will be used. Considerable laboratory testing appears to have demonstrated its successfulness in both monkeys and in human beings. He has at present immunized 700 persons with no untoward effects. He will immunize an additional 5,000 to 10,000 persons before January and before the National Foundation will start its program.

The vaccine is prepared from live virus killed by heat. It appears to be highly protective against Strain I and effective also, if perhaps to a slightly less degree, to Strains II and III. The immunization procedure shall consist of three injections of equal amounts, the first two spaced one week apart and the third consisting of a booster given four weeks after the second.

During the discussion, which continued for two and one-half hours, two principal subjects were discussed. The first of these concerned effectiveness and was quickly disposed of because the answer can be determined only after the completion of this national field trial. It appears effective in the very small number of instances in which the vaccine has been used at the present time. But no conclusions can be reached in that regard until many more persons have been included. The field trial is being performed for the purpose of determining the value of this procedure. It may, therefore, be some years before definite conclusions can be obtained.

The second problem of major interest concerned safety. Here also the evidence is presumed to be satisfactory, but the final determination may be obtained only after many more persons have been immunized. Every known precaution will be taken. Immediate and constant reporting will be required, and at the first indication of an untoward result the project will be cancelled across the nation. There have been no accidents or complications reported to date either in animals or among human beings. Each batch of vaccine will be tested on a minimum of 10 monkeys and in succession through 60 cultures to establish its purity and the fact that the virus has been killed. Every precautionary measure shall be taken and, as stated before, if any accident occurs, the project will be stopped.

The National Foundation will conduct the experiment and will assume responsibility attendant to its procedures. The National Foundation will select the areas in which the experiment is to be conducted. None will be selected on the basis of local requests, but only following the decision of the foundation. However, the test will not be conducted in any locality unless approval has first been obtained from the state medical society and the medical society of the county involved. The National Foundation will supply the vaccine to the state health officer, who will then transmit it to the medical profession within the area where it is to be used. The National Foundation will not supply adjunct materials and will pay no remuneration to physicians or other persons involved.

Moreover, the entire nation-wide test must be completed before June 1. The project will begin in January in the South, where the annual polio season begins early, and will proceed northward. Since the final injection must have been given before June 1, the project must have been started about the middle of May or preferably earlier.

The National Foundation has limited the test to second grade children. Children in the first and third grades will be analyzed for controls. In addition to approval from the medical profession, there must be a written signed request from the parents of each child to be immunized, and certain other records must be kept. Counties selected will probably represent a population of 50,000 or more, and in this state Johnson, Reno, Sedgwick, and Shawnee counties are under consideration. However, this may be altered, but again the decision rests with the National Foundation.

At the conclusion of this meeting, the Committee on Child Welfare further discussed the project and

took action declaring that they consider the procedure to be relatively safe, at least to the degree that they are willing to co-operate with the National Foundation. The action of this committee was then submitted to the Council of the Kansas Medical Society, which body also voiced its approval.

Letters have since been written to each county medical society in this state, requesting local action on this subject regarding the course to be followed should they be invited to participate. This was requested even of the smaller counties which undoubtedly will not be included in the first field trials, but it was felt that each county society should be given an opportunity for expression on this subject so that their opinions would be available if needed. It may be that this may ultimately be discarded. It may be found to have insufficient protective power. It may ultimately be discovered to be dangerous, but it could also be that December 18 may represent a landmark of historic importance in the record of medicine in Kansas.

VA Non-Service Connected Cases

Differences between organized medicine and the Veterans Administration are almost all centered around the disposition of non-service connected cases. The A.M.A. has taken action on this subject and in June, 1953, recommended that medical care of veterans should be limited to:

"1. Veterans with peacetime or wartime service whose disabilities or diseases are service-incurred or aggravated, and

"2. Within the limits of existing facilities, to veterans with wartime service suffering from tuberculosis or psychiatric or neurological diseases of non-service origin, who are unable to defray the expense of necessary hospitalization."

The report further states: "... the provision of medical care and hospitalization in Veterans Administration hospitals for the remaining group of veterans with non-service connected disabilities (should) be discontinued and the responsibility for care of such veterans revert to the individual and the community, where it rightfully belongs."

It appears that non-service connected cases currently represent 85 per cent of all patients discharged from VA hospitals, and that number is growing larger because of the continued liberalization of the VA's interpretation of service connection. When the veteran's condition is once presumed to be service connected, the veteran usually is entitled to the same benefits as one who was wounded in battle. He receives hospitalization, out-patient care, or home town care at the expense of the government and is eligible for financial compensation if his disability is found to be 10 per cent or more.

The veteran whose disability is non-service connected may receive free care only in a veterans' hospital and only if he signs the much publicized pauper's oath. Actually, that is a misnomer because, until recently, he has been required to state merely that he cannot afford to pay for his care. That statement is accepted at its face value and, until now, has not been questioned.

The Congress of the United States, as well as the VA, has repeatedly broadened presumption of service connection, each time adding to the ever growing list of veterans additional persons who may receive medical service at government expense, either in or outside a VA hospital. In 1921, Congress authorized service presumption for all tuberculosis and psychosis cases diagnosed within two years after discharge from service. In 1923, the Congress extended this from two years to three. In 1924, claims for active tuberculosis were considered conclusive and not subject to challenge by the government. A considerable number of other conditions was added to the presumptive list. There followed a brief period in which this was somewhat restricted, but by 1934 these benefits were all reinstated.

By 1948, 23 chronic diseases were specifically listed, and 16 additional tropical diseases were added that had never appeared before. And the VA Administrator was authorized to add such other diseases as he believed necessary. At that time, diagnosis had to be made within a one-year period. In 1950, this was extended to allow a three-year presumptive period for pulmonary tuberculosis. In 1951, multiple sclerosis was extended to two years. In 1953, all types of tuberculosis were extended to three years. In other words, if the disease was diagnosed within three years following the veteran's discharge from service, it was presumed to be service connected.

But all this appears only to be the beginning. Today there are many diseases that shall be presumed service connected if manifest within a year after separation from service. These include malignant tumors, diabetes, epilepsy, nephritis, calculi of the kidney, bladder, or gallbladder, many other conditions, "and such other chronic diseases as the Administrator of Veterans' Affairs may add to the list." Then there are 16 tropical diseases which are presumed service-connected, not only directly but also including any condition or disorder resulting from or originating because of therapy administered in connection with such diseases. The situation goes still further because the law states, "Nothing in this paragraph shall be construed to prevent service connection for any disease or disorder otherwise shown by sound judgment to have been incurred in or aggravated by active service."

The future portends still more. There are a number of bills previously introduced which will await action of the Congress when it reconvenes. Each

would liberalize the presumption period for a particular disease when the disability is 10 per cent or more. One would extend the presumptive period for all chronic and tropical diseases from one to three years. Another extends multiple sclerosis or psychoses to three years. Another extends the period for malignant tumors from one to two years. Another extends the three-year period for tuberculosis to seven years following separation. Still another would make the three-year period for active pulmonary tuberculosis conclusively presumptive so that it could not be contested by the government.

Perhaps there has been some result achieved in the continual opposition to this growing procedure. At least as of November 4, the VA has altered the application form for hospital treatment. Now, in addition to the statement of whether or not the veteran believes he can afford the cost of his care, he is asked to list the value of his property, the current amount of his assets, his average income, and his average expenditures.

On the same date, a letter explaining the use of the new form was sent out by the Administrator of Veterans' Affairs. This circular, Number 11, lists 11 points among which the following might be of interest to physicians. No investigation of any case may be made "except as authorized by Central Office." It states that information on this form is confidential and may not be given to any unauthorized person. Veterans who have been adjudicated to have service connected disabilities will not be required to fill in the addendum. Of more interest, however, are statements 3 and 4 which read as follows:

"3. This addendum may be used in no way whatever to deny hospitalization to a veteran as the law specifically provides that 'the statement under oath of the applicant...shall be accepted as sufficient evidence of inability to defray necessary expenses.'

"4. If a veteran-applicant for hospitalization signs the oath of inability to pay contained in VA Form 10-P-10, that is legal evidence of eligibility for hospitalization and the applicant shall be admitted when a bed is available and the need for hospitalization has been medically determined."

The Facts of Life...

Often times physicians are confronted with a problem of suggesting literature on a particular health problem to the patient. Louis I. Dublin, Ph.D., second vice-president of the Metropolitan Life Insurance Company, has written a condensed question and answer report on many phases of health as well as a wide range of facts relating to man's welfare. The book is entitled "The Facts of Life from Birth to Death."

Each chapter relates to a particular subject such as Population, The Pattern of Marriage, The Sick and Their Care, Cancer, Tuberculosis, Diabetes, other diseases of public interest, etc. The author has expertly chosen his words, and the language throughout the book is understandable to the average lay person.

If one reads an entire chapter, he would find himself well informed on at least the main points of a specific topic, and for those who wish to further their study a selected reference is given at the end of the volume.

Many physicians throughout the state are using the book in their reception rooms and recommending its use to lay persons. The interesting facts and statistics are a result of many years of research by the Metropolitan Life Insurance Company, which regularly makes a substantial contribution to the health of the nation through its services to policyholders, its publications, and its advertisements.

Change in A.M.A. Dues Policy

The House of Delegates of the American Medical Association, during the 1953 midwinter meeting in St. Louis, adopted a significant change in policy regarding the payment of dues. This may affect physicians who have not paid their dues for 1950. It is described in some detail because many Kansas physicians have not elected to become active members of the A.M.A.

This fact is of some interest to the Kansas Medical Society, first because the A.M.A. is the parent body which deserves the support of the members in this state and, second, because delegates to the A.M.A. are apportioned according to the number of members of the A.M.A. rather than on the basis of the number of physicians practicing within the state. Each state is afforded one delegate to the A.M.A. for each 1,000 members of that body.

In 1950 the A.M.A. voted to assess each member annual dues of $25. In that action was included a statement that subsequent dues for any physician who was a member of a state medical society in 1950 would not be accepted until the dues for 1950 had been paid. This has resulted in considerable confusion because many doctors have recently determined to join the A.M.A. but have been reluctant to pay that extra year's dues.

The current action of the House of Delegates temporarily changes this requirement. For a period of six months, an eligible physician may join the A.M.A. upon the payment of $25 without the payment of the dues for the year 1950. This applies only to physicians who join prior to July 1, 1954, and will not be continued after that time. Therefore, it is recommended to those members of the Kansas Medical Society who have previously not joined the A.M.A. and who wish to affiliate with that body,

that they send their dues before July 1. An additional advantage will result from such action because the A.M.A. is planning to publish a new directory and will distinguish the names of members from those who do not belong to the A.M.A.

The procedure for paying these dues is relatively simple and may be accomplished without unnecessary delay according to the following formula. A check for $25 made payable to the American Medical Association should be submitted to the secretary of the county medical society to which the physician belongs. This will be recorded and the check will then be transmitted to the Kansas Medical Society. Here also a record of the payment will be kept and the check will then be forwarded to the A.M.A. In due time a membership card will be received. This card will identify the holder as a member in good standing for the year 1954 and will entitle him to attend the meetings of the A.M.A. Already defined as to location are the annual meeting of the A.M.A. which will be held in San Francisco, June 21-25, 1954, and the midwinter meeting to be held in Miami early in December.

Certain physicians are entitled to membership within the A.M.A. without the necessity of paying dues. These exemptions must have been certified by the county medical society and by the Kansas Medical Society, but do not necessarily coincide with the exemptions of dues that pertain to the county and the state societies. In other words, not all honorary members of the Kansas Medical Society are exempt from the payment of A.M.A. dues. However, none will be exempt except upon certification from the local society. The A.M.A. has established six categories for exemption which are listed by code as follows: Group A, exempted by reason of serving a residency; Group B, on the basis of financial hardship; Group C, those in military service; Group D, disability; Group K, permanent retirement, and Group L, all physicians who are 70 years of age or older.

Again, the A.M.A. will not accept the word of the physician that he is in military service, for example. This must be certified by the county and the state medical societies. It is recommended, however, that the individual physician take the initiative for requesting such exemption and report this request to the secretary of his county society. The same applies to members who reach 70 years of age. Here the exemption becomes automatic, but only after certification. It is not sufficient for the physician to inform the A.M.A. of this fact. It must be accomplished by letters from the county and state societies. Here again, the Kansas societies will not immediately be aware of these facts, and it is urged that the individual physician make his request for exemption if, in his opinion, he might qualify under any of the six categories.

The Kansas Medical Society hopes that each member will unite with the A.M.A. This is not mandatory and has no bearing on the individual's status with either his county or the state society. It is important to everyone that all physicians belong to the A.M.A., and this special notice is hereby given because of the temporary change of policy which may provide an inducement for a considerable number of physicians to join at this time.

Annual Meeting, May 2-6, 1954

Although the complete program for the 1954 annual meeting of the Kansas Medical Society, to be held at Topeka, May 2-6, 1954, cannot be announced at this time, plans are rapidly being completed for the occasion. Committees of the Shawnee County Medical Society, host for the session, are meeting regularly to work out details.

In general, the plan of former sessions will be followed. Scientific programs will begin on Tuesday, May 4, and continue through the morning on Thursday, May 6. The annual banquet will be held, as usual, on Wednesday evening. The first meeting of the House of Delegates will follow a dinner on Tuesday evening, and the second meeting will follow a luncheon Thursday noon.

A chronological listing of events includes two meetings on Sunday, May 2. First is the annual meeting of the Board of Directors of Kansas Physicians' Service (Blue Shield). The second Sunday event is the opening of the two-day meeting of the Kansas Medical Assistants' Society.

There will also be two events on Monday, May 3, in addition to a continuation of the medical assistants' program. One is the annual meeting of the Kansas Academy of General Practice, including both scientific and social programs and concluding with a banquet. Of primary interest to sportsmen is a golf and skeet tournament during the day and a tournament banquet at the Topeka Country Club in the evening.

The Woman's Auxiliary to the Kansas Medical Society will hold its annual meeting on the days of the scientific sessions. A number of specialty societies are also planning sessions during the week.

It is recommended that all who plan to attend make hotel reservations now. Rooms are still available at the two major hotels, and reservations may be made through Mr. Harry Lose, manager of the Jayhawk, and Mr. Earl Shore, manager of the Kansan. Motel accommodations may be secured at the Jayhawk Junior, Meadow Acres, Nod-Away courts, and others.

K.M.S. annual meeting, May 2-6, 1954, Topeka.

Adenocarcinoma of the Prostate

Tumor Conference

Edited by Chauncey G. Bly, M.D., and Irwin Joffe, M.D.

Dr. Helwig: Today we have two patients with prostatic cancer; one is without demonstrable metastases or evidence of extension; the other is a relatively young man for cancer of the prostate, and he shows extensive metastases. I would like Dr. Shinkle to give us the essential facts in the history of the first patient.

CASE NO. 53-74

Dr. Shinkle: This 69-year-old white man had had symptoms of prostatism for three years; during the 9 months prior to admission he had noted increased dysuria and frequency. On admission, an enlarged (two-plus), nodular, stony-hard prostate was palpated on rectal examination. The serum levels of acid and alkaline phosphatase were normal. One week after admission, the patient underwent transurethral prostatic resection, and a few days later bilateral orchiectomy was done. He returned home on the 16th hospital day.

Dr. Helwig: I understand that this patient had no significant x-ray findings. May we have the pathologist's discussion?

Dr. Boley: Only a few of the many fragments of prostate tissue submitted show areas of tumor tissue. This relative paucity of tumor tissue in the large sample examined would indicate that either this patient has a rather small carcinoma, or that the region of prostate biopsied contains less tumor than do other regions.

Often as one approaches these areas of carcinoma, he sees a marked tendency for infolding and proliferation of the epithelium in the remaining glands of the prostate (Figure 1). Supporting stroma is completely lacking in these papillary epithelial folds. In some cases, when a pathologist sees only these peculiar proliferations within prostatic acini, so-called "glands within glands," he becomes suspicious, studies further sections and, as in this case, very often turns up one or more foci of unequivocal carcinoma.

CASE NO. 53-75

Dr. Shinkle: The second patient, a 57-year-old white man, was first admitted 9 months ago, after urinary retention had required constant catheter drainage for 5 days at home. He at that time gave a history of "prostatism" of rather short duration, approximately 4 months. The patient walked with a limp, with obvious pain on movement of his left leg. Rectal examination revealed a slightly (one-plus) enlarged smooth, hard prostate. Pertinent laboratory findings included a serum acid phosphatase level of 0.6 millimols (normal 0.1-0.6) and an alkaline phosphatase level of 7.1 millimols (normal 0.7-3.0). After a diagnosis of carcinoma of the prostate had been made on tissue removed by transurethral resection, a bilateral orchiectomy was also recommended, which the patient refused at that time. He was placed on 5 mg. of stilbestrol and dismissed.

Approximately one month later his family physician did perform a bilateral orchiectomy. The patient was seen periodically here in the clinic during the next 7 months. Three days ago he returned to the clinic with increasingly severe pain in his lower back and legs, evidence of extensive metastases. He entered the hospital for further evaluation and for consider-

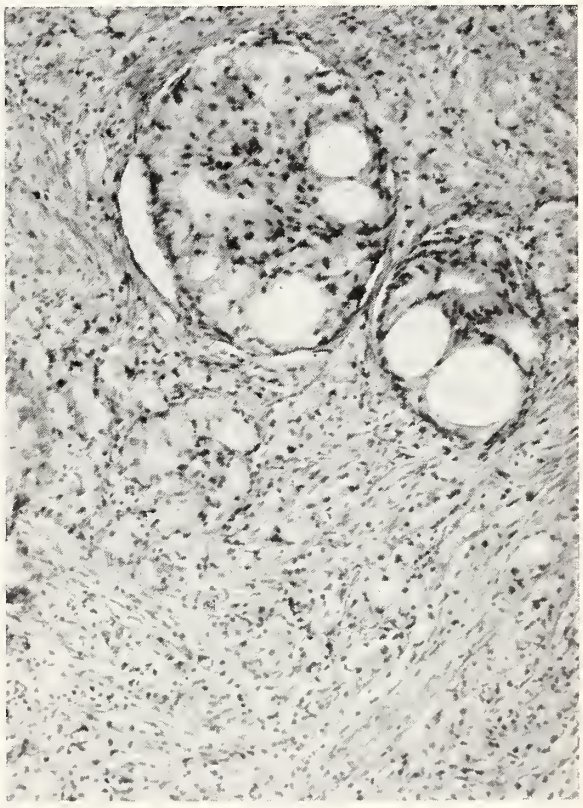


Figure 1. Atypical proliferation of glandular epithelium within two prostatic acini, which are surrounded by sheets of partially differentiated carcinoma cells invading through the prostatic stroma. X 120. Hematoxylin and eosin.

Cancer teaching activities at the University of Kansas Medical Center aided by grants from the National Cancer Institute and the Kansas Division of the American Cancer Society.

Dr. Bly is a scholar in cancer research of the American Cancer Society. Dr. Joffe is a trainee of the National Cancer Institute.

ation of the advisability of therapy with cortisone.

Dr. Litton: Serial roentgenograms of the chest and kidney-ureter-bladder tract during the last 9 months show progression of extensive osteoblastic metastases in the ribs, pelvic bones, and especially in the dorsal spine (Figure 2).

Dr. Helwig: Did examination of the surgical specimens from this case reveal anything different from those of the first case, Dr. Boley, and would you be able to make any predictions from your studies?

Dr. Boley: Microscopic sections from representative fragments of this patient's prostate show moderate numbers of dilated and hyperplastic glands. Much more important, however, are the nests of atypical epithelial cells interposed in great numbers between bands of fibrous and muscular stroma. These epithelial cells vary much more widely in size and shape than do the cells of the typical adenocarcinoma of the prostate as illustrated in the first case. The poorly differentiated or anaplastic tumor cells appear incapable of forming recognizable glands or acini in this second case.

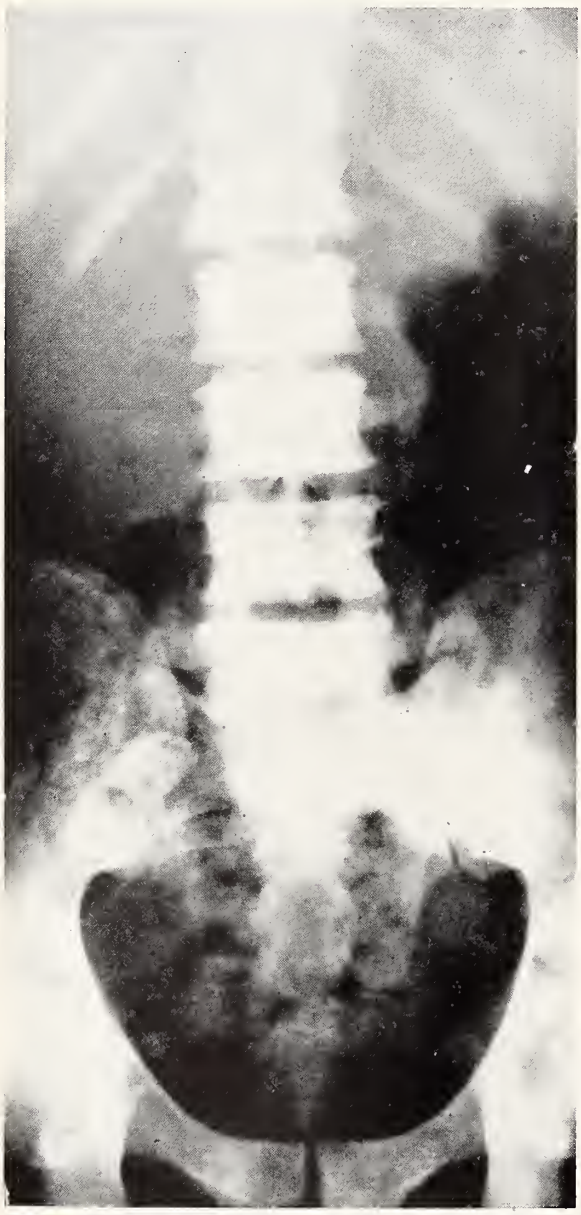


Figure 2. Roentgenogram showing chalky irregular areas of increased density indicating extensive osteoblastic metastases to ribs, vertebrae, and pelvic bones.

The histologic appearance of the tumor cells and glands in carcinoma of the prostate is of little value in estimating duration, present size, or extension of the tumor, or of predicting future potentialities. Even though, in today's cases, there were no demonstrable metastases of the better differentiated carcinoma, and disseminated metastases from the anaplastic one, we still hesitate to prognosticate on the basis of the histologic appearance of cancer of the prostate. Most pathologists today feel that there is little to be gained by struggling to classify carcinomas of the prostate according to the degree of differentiation or maturation of the tumor cells. For example, Foot[1] found no differences in prognosis in patients with diffusely infiltrating anaplastic tumors as opposed to those with well-differentiated carcinoma.

Dr. Helwig: There are several other important areas of discussion in today's topic. Would you lead off, Dr. Valk, by defining prostatism for us?

Dr. Valk: By prostatism we mean (1) increased frequency of urination, (2) decrease in size and force of the urinary stream, and (3) increased effort in voiding. The term comes from the fact that these signs and symptoms are often related to the strategic location of the prostate around the urethra and neck of the bladder. Prostatism occurs in about one quarter of the male population over the age of 55. We feel that it is probably quite normal for a man over the age of 50 to void once at night, but if he gets up more often than this, it is a definite sign of partial obstruction at the bladder neck or of disease of the nerves supplying the bladder. Increased frequency of urination, in the absence of infection or foreign bodies such as stones in the bladder, usually occurs because of failure to empty the bladder at each voiding. With obstruction at the neck of the bladder, the bladder muscle first hypertrophies in an attempt to expel all the urine through the diminished orifice. As long as the increased muscle mass and contractile power can compensate for the partial obstruction, there will be little residual or unexpelled urine. Sooner or later, however, the bladder muscle becomes decompensated, and residual urine will be left in the bladder after voiding.

Three pathological entities commonly cause prostatism: benign prostatic hypertrophy and hyperplasia; adenocarcinoma of the prostate; and vesicle neck contracture.

Approximately 70 per cent of the patients with prostatism have benign prostatic hypertrophy and hyperplasia. The actual cause of this overgrowth of the smooth muscle and the glandular elements in the prostate is not known. However, it is reasonable to believe that variations in the endocrine equilibrium are responsible for the overgrowth, since the prostate in growth, health, or disease is sensitive to endocrine factors.[2] At puberty, the anterior lobe of the pituitary gland produces luteinizing hormone which stimulates the interstitial cells of the testis to produce male sex hormone. The male sex hormone in turn stimulates the prostate to increase in size and to produce secretion. Another pituitary principle, prolactin, apparently helps to mediate these effects.[3] There is very little change in the prostate gland from puberty until about the age of 50 when, most probably because of changes in the pituitary, adrenal glands, and testes, about 25 per cent of the male population develop further overgrowth of the prostate.

It is interesting to note that if a young man is given an excessive amount of anterior pituitary preparation, prostatic hypertrophy will often develop. We have seen pre-pubertal boys with undescended testes who had received excessive amounts of anterior pituitary preparation; all of them had a definite increase in the size of the prostate, which in one of these little boys produced urethral obstruction.

The Sertoli cells of the testes, and the adrenal cortex, probably produce estrogen-like hormones, and as a man grows older there seems to be gradually increased production of these female substances. On the other hand, the male sex hormone, produced by the Leydig cells of the testes, shows an increase in production from the time of puberty to about the 28th year, and thereafter begins a gradual decline. Many people believe that the etiology of hypertrophy of the prostate is somehow bound up with this shift in the androgen-estrogen balance.

Carcinoma of the prostate is the second lesion responsible for prostatism and is the second most common cancer in men over 55 years of age. Approximately 20 per cent of the patients seen in this clinic with prostatism have detectable carcinoma of the prostate. However, only a very small percentage of this group have small isolated nodules of carcinoma which can be satisfactorily treated by removal of the entire prostate and seminal vesicles.

The third lesion responsible for prostatism is contracture of the vesicle neck. This disease also has an obscure etiology. We formerly believed that the scarring at the neck of the bladder was due to previous inflammation and infection in the prostate. Obviously this is not true, since the tissue that we remove in these cases consists mainly of hypertrophied smooth muscle from the neck of the bladder. This lesion is perhaps comparable to the hypertrophy of smooth muscle that is seen in pyloric stenosis in children.

Dr. Helwig: Dr. Valk, would you next discuss digital examination of the prostate through the rectum, and instruct us regarding the clinical grading of the degrees of enlargement?

Dr. Valk: The adult prostate is primarily composed of two lateral lobes and one median lobe. The lateral lobes meet to form a posterior central longitudinal sulcus which is palpable through the anterior rectal wall. A normal prostate is roughly the size of a horse chestnut (3 to 4 cm. in diameter) and weighs from 15 to 28 grams. If the prostate is felt to be slightly larger than the gland just described, it is called "one-plus" enlarged. If the gland is so large that the average examining finger won't go over the top of the prostate and can't reach to the lateral margins, we say the prostate is "four-plus" enlarged. If the finger goes just over the top of the gland and just around the sides, we call it "three-plus" enlargement; and if it's somewhere between one and three, we call it "two-plus." It is difficult to arrive at more exact dimensions by digital palpation through the rectum.

However, the size of the prostate as judged by rectal palpation is not the most important physical characteristic, for the external size and shape may have nothing to do with the symptoms produced. One patient with a small prostate may have severe urethral obstruction due to marked proliferation of periurethral glands only, whereas another individual may have a prostate the size of a grapefruit (9-10 cm.) with practically no urethral obstruction. Furthermore, today's patient with the smaller prostate had many metastases from the carcinoma there.

The most important characteristic of the prostate to be always evaluated clinically is the consistency, which normally is about as soft as the end of one's nose. Any prostate which has focal hard areas or is diffusely hard must be considered to be carcinoma of the prostate until proved otherwise by adequate biopsy. Carcinoma of the prostate, like palpable carcinoma anywhere, ought to be diagnosed earlier than it is. Unfortunately, we see annually fewer than five patients with localized prostatic carcinoma on whom we can confidently attempt a radical operation for removal of the entire prostate and seminal vesicles. If an entire lobe is clinically involved, we usually expect the pathologist to find that cancer has already spread to the seminal vesicles and the regional lymphatics.

Dr. Helwig: Pathologists grade prostatic carcinoma in the following manner.[4] If the cancer is confined entirely to the prostate, it's "Grade One"; when it invades the capsule, it's "Grade Two"; when it reaches the seminal vesicles, it's "Grade Three"; and when it extends further into regional

lymphatics or when it metastasizes to more distant sites, it's "Grade Four." As Dr. Valk mentioned, the only ones that are amenable to surgery are those which we would call "Grade One." These unfortunately constitute less than 5 per cent of the clinical cases of cancer of the prostate.

It is worth mentioning that a year or so ago Hudson[4] examined a group of 59 indigent elderly gentlemen who lived in a Bowery hotel in New York and found that 30 per cent of this group had evidence of cancer of the prostate. This study offers some indication of the remarkable frequency of prostatic cancer in advancing age.

In regard to liaison between the pathologist and the clinician who are attempting to reach a diagnosis on an abnormal prostate, I use what I call the "rule of finger": "If the urologist's finger says that it is carcinoma and the microscope says it's not carcinoma, *it's carcinoma;* if the urologist's finger says it's not carcinoma and the microscope says it is carcinoma, *it's carcinoma.*"

Because so frequently a carcinoma starts in the posterior lobe, it has to extend for a considerable distance before it obstructs the urethra. Moreover, when patients have both benign hyperplasia and carcinoma, as they frequently do, the obstructing element in the prostate may be the benign hyperplastic tissue and not the cancer at all. Therefore, when the urologist relieves such an obstruction with the resectoscope, only a few of the deeper fragments removed may contain carcinomatous tissue, as in today's first case. It is impractical to make serial sections of every fragment received in all cases. Thus, when the pathologist examines what he considers a fair and representative sample of the material, he may miss the areas of cancer entirely. I personally do not hesitate to ask the urologist what his digital examination of the prostate disclosed. If he strongly suspects carcinoma, I section additional material and occasionally will find in one or two fragments a miniature focus of cancer. The urologists understand our problem and inform us of their clinical suspicions.

Now, I would like to have Dr. Valk review for us other aspects of the management of patients with carcinoma of the prostate.

Dr. Valk: In 1941, Dr. Huggins[5] felt that, since the male sex hormone was responsible for the development and maintenance of the prostate, patients with carcinoma of the prostate might benefit by removal of the testes. He was gratified to find that after orchiectomy about 75 to 80 per cent of patients with far advanced carcinoma of the prostate went into remission. At about the same time, it was discovered by Dr. Herbst[6] that orally administered stilbestrol effectively neutralized or inactivated male sex hormones and induced remissions in a similar proportion of patients. Dr. Phil Clark[7] also had been working on endocrinological problems in Dr. Huggins' laboratory and had already discovered that, in this apparent neutralization or inactivation, the female sex hormone was found to be about 10 times as potent as the male sex hormone. People have known for a long time that the female influence was something to be respected, but here was laboratory proof! When we give a male patient 5 mg. of stilbestrol daily, we give him many times more female sex hormone than is necessary to cause changes in the endocrine system that approximate castration.

In 1946, Nesbit and Plumb[8] studied 1,800 patients who had had bilateral orchiectomy and estrogen therapy, and it was discovered that usually within three years and not longer than five years after castration, the patients relapsed. Moreover, the percentage of five-year survivals in the group receiving orchiectomy or stilbestrol therapy or both was no higher than in the untreated group. One explanation that had been previously proposed suggested that the adrenal cortex, behaving like a third testicle, took over some of the function of the testis and produced enough androgenic steroid material to cause reactivation of the cancer. In fact, in 1943-1944, before the days of cortisone, bilateral adrenalectomy had been carried out on three patients, but since at that time adrenal replacement therapy was very difficult, the results could not be accurately appraised.[9]

Finally, with the advent of cortisone, bilateral surgical adrenalectomy has now been performed in about 200 cases of carcinoma of the prostate in this country. Most of the statistics available indicate that this procedure does give palliation in from one-fourth to one-half[10] of the cases but that most of the patients died within 4 to 9 months after adrenalectomy. An even more heroic operation, hypophysectomy, has also been recently tried in a few patients, with too few cases to give unequivocal results.[3]

It has been known for a long time that cortical atrophy and decrease in adrenal cortical function occur following prolonged cortisone therapy. We have recently treated 15 patients with orchiectomy followed by estrogen and cortisone. All of these patients had had far advanced carcinoma of the prostate with many metastases, and had relapsed after first improving on castration therapy. It appears to us at present that the maximum inhibition of far advanced carcinoma of the prostate may be best obtained by a combination of orchiectomy, 5 mg. of stilbestrol a day, and maintenance doses of cortisone.

Dr. Helwig: Have you tried progesterone or other steroids?

Dr. Valk: No, we haven't. Some physicians have tried progesterone and others but felt that the temporary improvement noted was too slight to warrant continued study.[11]

I doubt that the effects of administered estrogens can be satisfactorily explained by a strictly chemical concept of neutralization of male sex hormones. Pos-

sibly the estrogen works upon some such organ as the pituitary, resulting in a depression of the production of gonadotrophic hormones.

Dr. Helwig: I can recall one attempt to explain the effect of administered estrogens, on a more anatomical basis. These investigators[12] noted fibrous sclerosis of the prostate in castrated guinea pigs which had been given estrogens, and they suggest the possibility that this sclerosis might be a direct effect of the estrogen on the prostate.

On the other hand, physiologists have for a number of years speculated as to the ability of the normal body to convert androgen to estrogen or the reverse. Recently a group of investigators[10] at Harvard have found that administered androgens are converted into estrogens in the male, and excreted as such in the urine.

Dr. Bly: Along these lines of thought, I recall that one of the best sources of the original estrone was stallion urine, which may contain 100 times as much estrogenic material as even human pregnancy urine. Strangely enough, the testis of the horse is said to be the richest known tissue source of estrogens,[13] but this does not prove for us that androgens are converted into estrogens in the testis or in any other organ of the body. Certainly such conversion can be done in the chemical laboratory.

Dr. Helwig: Isn't it true that less than one-half of the small localized curable prostatic carcinomas can be diagnosed preoperatively?

Dr. Valk: Yes, I agree. Moreover, these small cancers of the prostate pose difficult problems in management too. A person in his 50's with a small nodule in the prostate is an example of the typical problem-case. We feel that the nodule ought to be removed or biopsied. Sometimes we will use the Turkel needle to obtain a piece of that nodule. However, it is occasionally very difficult, even with one's finger in the rectum, to insert a needle into such a nodule; therefore, a negative result means nothing.

On the other hand, if we are compelled to explore thoroughly the prostatic capsule, we render the patient impotent. Since only about one-half of these nodules are malignant lesions, we must weigh the evidence carefully before proceeding to destroy the nerves of the posterior capsule. Many of these nodules prove to be only fibromas, healed inflammatory areas, prostatic abscesses, or calculi. Consequently, we discuss these matters thoroughly with the patient and explain that sometimes the search for cancer involves the loss of his sexual powers.

Dr. Helwig: I heard Dr. Flocks[14] give his paper at Denver this summer; he had given radioisotopic colloidal gold to 160 patients with carcinoma of the prostate. Can you elaborate on some of his studies for us, Dr. Valk?

Dr. Valk: Dr. Flocks wanted to work out a better method to control far-advanced infiltrating cancer of the prostate. When radioactive gold (Au^{198}) became available, it was discovered rather early that this material, when injected, followed the lymphatic drainage pattern of the area and could often be picked up in the regional lymph nodes and lymphatic channels. Dr. Flocks opens the bladder widely through a suprapubic incision and injects colloidal gold directly into the carcinoma of the prostate. There are many complications to this type of irradiation therapy. However, it often does cause quick regression of the local tumor masses and may even temporarily suppress growth of extensions of the tumor along lymphatics. It offers a method of palliative irradiation of the tumor, with less damage to overlying tissue than x-irradiation would produce. It is possible that one might cure patients of small localized cancers within the prostate using this method, but surgery seems to offer as good or better chances for cures in these cases.[3]

Dr. Helwig: Dr. Tice, would you like to comment on the irradiation treatment of advanced prostatic carcinoma?

Dr. Tice: The only cases I recall in which radiation has been of benefit have been those with bony metastases where pain was a predominant symptom. I am not impressed by a great amount of ultimate benefit to be derived from irradiation in cancer of the prostate, but definite relief of bone pain can be obtained in certain cases with irradiation directly over the metastatic sites.

Dr. Helwig: Has anyone done any bone marrow studies at this institution on early prostatic carcinoma? I recall a study of 60 cases of early carcinoma of the prostate reported a year or so ago.[15] The authors found positive marrow smears in 19 cases, 5 of which showed no evidence of x-ray changes. It was suggested by this group that many of those patients without the x-ray findings or alterations in serum acid or alkaline phosphatase levels might already have metastases that could be discovered by marrow biopsy. The procedure was offered as another diagnostic adjunct to the study of the patient with carcinoma of the prostate before doing radical surgery. Both ilium and sternum were used as biopsy sites with equivalent results. Such studies might be appropriate in our first patient for study today, if he returns to the hospital.

Dr. Tice: I wonder if anyone would comment on the relative infrequency of lung metastases in carcinoma of the prostate, despite metastatic lesions in adjacent ribs.

Dr. Helwig: Pulmonary metastases are relatively uncommon, whereas I suspect that, if we did complete skeletal surveys on all autopsied cases of carcinoma of the prostate, we would find some bone metastases in an extraordinarily high percentage of cases, maybe up to 90 per cent. In Kaufmann's book[16] mere routine skeletal studies disclosed bony

metastases in 50 per cent of cases dying of carcinoma of the prostate. Perhaps the figures would be lower today with better and earlier diagnosis and treatment.

BIBLIOGRAPHY

1. Foot, N. C.; Humphreys, G. A., and Coats, E. C.: Carcinoma of the prostate, N. Y. State J. Med. 50:84-90, 1950.
2. Scott, W. W.: What makes the prostate grow, J. Urol. 70:477-488, 1953.
3. Scott, W. W.: Reflections on the endocrine management of disseminated prostatic cancer, special article for Year Book of Pathology, Year Book Publishers, Chicago, 1952 (pages 1-14; also editorial comments on pages 195, 291).
4. Hudson, P. B.: Diagnosis of prostatic cancer, Cancer Bulletin 4:41-42 (March) 1952.
5. Huggins, C. B., and Hodges, C. V.: Studies on prostatic cancer: I. Effect of castration, and of estrogen and androgen injections on serum phosphatases in metastatic cancer of the prostate, Cancer Research 1:293-297, 1941.
6. Herbst, W. P.: The present picture in chemotherapy in carcinoma of the prostate, J. Urol. 57:296-299, 1947.
7. Huggins, C. B., and Clark, P. J.: Quantitative studies of prostatic secretion: effects of castration and estrogen on prostate glands of dogs, J. Exp. Med. 72:747-762, 1940.
8. Nesbit, R. M., and Plumb, R. T.: Prostatic carcinoma, Surgery 20:263-272, 1946.
9. Butler, W. W. S.; Grayhack, J. T.; Ransom, C. L., and Scott, W. W.: Metabolic studies on the bilateral adrenalectomy patient, J. Urol. 70:657-664, 1953.
10. Abstracts: New developments in cancer, CA, Bulletin of Cancer Progress 3:88 (March) 1953.
11. Brendler, H.: Evaluation of current treatment of prostatic cancer, J. Urol. 68:734-743, 1952.
12. Nicol, T.; Helmy, I. D., and Abou-Zikry, A.: Histologic explanation for the beneficial action of endocrine therapy in carcinoma of the prostate, Brit. J. Surgery 40:166-172, 1952.
13. Pincus, G., and Pearlman, W. H.: Intermediate metabolism of the sex hormones, Vitamins and Hormones 1:294-333, 1943.
14. Flocks, R. H.: Treatment of cancer of the prostate, Rocky Mountain Cancer Conference, Denver, 1953; also J. Urol. 70: 491-498, 1953.
15. Clifton, J. A.; Philipp, R. J.; Ludovic, E., and Fowler, W. M.: Bone marrow and carcinoma of the prostate, Am. J. Med. Sci. 224:121-130, 1952.
16. Kaufmann, E.: Lehrbuch der Speziellen Pathologischen Anatomie, Vol. I:957, Berlin und Leipzig, 1922.

A.M.A. House of Delegates

The House of Delegates of the American Medical Association, meeting at St. Louis, December 1-4, took important policy actions on social security, voluntary health insurance, medical ethics and unethical practices, medical education, hospital accreditation, military affairs, and a wide variety of subjects affecting both physicians and the public.

Highlight of the opening session was the announcement that Dr. Joseph I. Greenwell of New Haven, Kentucky, had been selected by a special committee of the A.M.A. Board of Trustees as the 1953 "General Practitioner of the Year." The annual medal and citation for community service by a family physician were presented to Dr. Greenwell by Dr. Edward J. McCormick of Toledo, Ohio, president of the American Medical Association, who also addressed the opening session.

The Tuesday program also included addresses by Dr. James R. Reuling of Bayside, New York, speaker of the House of Delegates, and Dr. Chester Keefer of Boston, special assistant to Mrs. Oveta Culp Hobby, United States Secretary of Health, Education and Welfare. Annual reports were presented by Dr. George F. Lull, secretary and general manager of the A.M.A.; Dr. Dwight H. Murray of Napa, California, chairman of the Board of Trustees, and by the standing and special committees of the House of Delegates.

Approving a recommendation by its Reference Committee on Legislation and Public Relations, the House passed a resolution reaffirming its opposition to the compulsory coverage of physicians under the Old Age and Survivors Insurance provisions of the Social Security Act and advocating passage of the Jenkins-Keogh bills now pending in Congress. These bills were described as providing for "the development of a voluntary pension program which is equitable, free from compulsion, and satisfies the retirement needs of physicians."

The reference committee report adopted by the House said: "The purpose of these bills is to eliminate the discrimination and inequities which exist under present tax laws by extending the tax deferment privilege to the country's 10 million self-employed and also to millions of employees who are not covered by pension plans. The purpose of the resolution is to reaffirm our support to the voluntary pension program provided in the Jenkins-Keogh bills and to reaffirm our strong opposition to the extension of compulsory coverage of physicians and other self-employed persons under Title II of the Social Security Act."

The same committee report urged continued action to obtain passage of the Bricker Amendment (S.J. Res. 1) and approved the principle of legislation which would reduce or remove the limitation on the deduction of medical and dental expenses for income tax purposes. It also opposed any further extension of the "Doctor Draft" Law beyond the present expiration date of June 30, 1955.

The report said that "your committee feels strongly that there should be no further extension of the 'Doctor Draft' Law. We feel that the legislation is discriminatory and urge the Committee on Legislation and the Board of Trustees to actively oppose any further extension."

The House acted to accelerate the development of voluntary health insurance by passing a resolution requesting the Council on Medical Service to proceed immediately with a special study of the problems of catastrophic coverage and coverage for retired persons. The Council was asked to present its findings and recommendations to the House not later than the 1954 clinical meeting. The resolution pointed out:

"There are two large groups of citizens for whom improved coverage could be offered under present prepaid medical care plans, namely: (a) those individuals who suffer catastrophic or long-continued and highly expensive illness and whose financial resources are not adequate to meet the cost thereof and (b) those citizens who have retired and are living

on small incomes and who are not eligible under presently existing public or private plans."

The resolution emphasized the medical profession's "responsibility to make every effort to promote such prepaid medical coverage for all citizens whose circumstances make them eligible."

Another resolution on voluntary health insurance, adjudged to be emergency business by the Reference Committee on Insurance and Medical Service and then passed by the House, stated that "the American Medical Association condemns all insurance contracts which classify any medical service as a hospital service." The resolution reaffirmed previous actions of the House defining pathology, radiology, anesthesiology, and physiatry as medical services.

A second emergency resolution, which would have endorsed the principle of federally subsidized scholarships for prospective military personnel in order to encourage the building up of a career-basis medical corps for the armed forces, was referred by the House to the Board of Trustees for study and action.

A resolution introduced by the Iowa State Medical Society, calling for approval of a joint-billing procedure involving services rendered by two or more physicians, was referred to the Judicial Council, at the suggestion of the Reference Committee on Miscellaneous Business, with the recommendation "that the Judicial Council investigate the factors involved in the matter as presented and determine if there are new factors or new facets that would cause it to change the opinion" determined in 1952.

The House approved a revision of one section of the Principles of Medical Ethics of the A.M.A. which clarifies the relationship of physicians to all forms of public information media. The revision had been worked out by the Council on Constitution and By-laws.

In an effort to solve the publicity problems resulting from unethical practices by a small minority of doctors, the House referred to the Board of Trustees a resolution calling for appointment of a special committee with broad professional representation to study all aspects of the problems. The board was asked to study and implement the intent of the resolution and to report its findings to the House at the June, 1954, meeting in San Francisco.

To clarify misunderstandings among physicians regarding the rules and regulations of the Joint Commission on Accreditation of Hospitals, especially as they concern the role of the Department of General Practice in a hospital, the House adopted the following resolution:

"That this House of Delegates of the American Medical Association request the Joint Commission on Accreditation of Hospitals to publish an article, or series of articles, in the *Journal of the American Medical Association* and other official publications circulating among the medical and hospital professions, to acquaint the medical-hospital profession with the regulations, by-laws and their interpretations, and

"That the Commission clarify the methods by which an aggrieved hospital or its staff may appeal a decision with which they are not in agreement."

In the field of medical education the House was "pleased to note" that a fourth grant of $500,000 had been made by the American Medical Association to the American Medical Education Foundation for financial aid to the nation's medical schools. The Foundation reported that its 1953 income now totals $1,174,000 and that the number of contributors now is more than double the total in 1952.

Dr. Louis H. Bauer, of New York, immediate past president of the A.M.A., was elected president of the foundation just prior to the opening of the A.M.A. clinical session. He succeeds the late Dr. Elmer L. Henderson of Louisville, also an A.M.A. past president.

At the opening session of the House, Dr. McCormick in his presidential address made a strong appeal to the nation's physicians for "action that will further the full confidence of the public in our profession."

"Good public opinion cannot be bought," he declared. "It must be earned through exemplary conduct and genuine service in the public interest. Whatever money the A.M.A. and its constituent societies spend for public education and public relations is wasted unless individual physicians take wholehearted interest in assuring the success of these ventures."

Dr. Reuling, emphasizing that much serious work remains to be done, warned that "times are just as troubled as when we had blanket bills before Congress which would have socialized the practice of medicine."

Dr. Keefer told the House that "the voluntary way has been the most successful in the past and there is no reason to believe it will not continue to be in the future." He urged maximum effort, co-operation and leadership on the community level.

Just prior to the clinical meeting the Joseph Goldberger award for outstanding contributions in the field of clinical nutrition was presented to Dr. James Somerville McLester of Birmingham, Alabama, a practicing physician for more than 50 years. The award was presented by the A.M.A. through its Council on Foods and Nutrition.

Final registration at the St. Louis Clinical Session was expected to total approximately 7,500, including about 2,700 physicians.

LAURENCE S. NELSON, M.D.
JOHN M. PORTER, M.D.
Delegates from Kansas

Magazine Approves A.M.A. Stand

An editorial upholding the stand of the A.M.A. on the subject of medical care for veterans with non-service-connected disabilities appeared in the October issue of *America*, a Jesuit magazine. A portion of the editorial is quoted below.

"Dr. Edward J. McCormick, president of the A.M.A., has repeatedly voiced opposition to this government largess. Behind the A.M.A. opposition lies the fear that socialized medicine may get into this country through the back door of the Veterans' Administration. Present veteran population is about 20 million and growing at the rate of one million a year. If free medical service in state-owned hospitals from state-salaried doctors becomes the rule, the government, the doctors claim, will soon control medicine. A.M.A. fears on this point seem well-founded. More than $500 million was spent on veterans' medical care last year, of which only $178 million went for the care of service-connected disabilities. Besides all this there is another big objection to the present law. The General Accounting Office, checking a test sample of 350 patients recently discharged from VA hospitals, found incomes ranging from $4,000 to $50,000. Twenty-five of these veterans had real property and other assets of from $20,000 to $500,000. Yet all had presumably sworn that they were unable to defray expenses. As an editorial in New York Medicine (August 20, 1953) remarks: 'Seldom has lying been so amply rewarded. Seldom has perjury been so widely condoned.' The A.M.A. deserves strong support on this issue."

A pure compound which temporarily lowers blood pressure in animals has been isolated from rhododendron leaves by scientists of the National Heart Institute. Chemically, the compound is pure, but its structure is a mystery. Called andromedotoxin, the substance resembles the veratrum alkaloids in physiological actions, but it is not nitrogen-containing, as alkaloids must be. It has a strong but brief hypotensive effect in low doses.

The drug has never been tested clinically, but it may have therapeutic possibilities. Although it will not be a cure for hypertension, it is felt that it may be found useful in lowering blood pressure in certain cases of hypertension.

Serves on A.M.A. Committee

Dr. John M. Porter, one of the Kansas delegates to American Medical Association meetings, served as chairman of the Committee on Insurance and Medical Service at the A.M.A. session in St. Louis last month. The committee had a number of important services to perform, and Dr. Porter received many commendations on his work with the group.

Dr. Theodore G. Klumpp, president of Winthrop-Stearns, Inc., pharmaceutical manufacturer, has been appointed a member of the Task Force on Medical Services of the Commission on Organization of the Executive Branch of the Government, under the chairmanship of former President Herbert Hoover. The commission, originally authorized by Congress in 1947, was recreated last July.

Objectives are to: provide better medical care for beneficiaries of the federal medical program, create a better foundation for training and medical service in federal agencies, reduce the drain of doctors away from private practice, provide better organization for medical research, and provide a better state of medical preparedness for war.

Eighty per cent of vehicles involved in fatal traffic accidents in 1952 were traveling straight ahead.

95th annual meeting, Kansas Medical Society, Topeka, May 2-6, 1954.

Patronize Journal advertisers.

DEATH NOTICES

Charles Frederick Menninger, M.D.

Dr. C. F. Menninger, 91, founder of the Menninger Foundation, died at his home in Topeka on November 28 after an illness of several weeks. He began practice in Topeka shortly after his graduation from Hahnemann Medical College and Hospital, Chicago, in 1889. His plan to found the Menninger Clinic was realized in 1919 when his son, Dr. Karl Menninger, returned to Topeka after serving a residency in neurology and psychiatry at the Boston Psychopathic Hospital. Another son, Dr. William C. Menninger, joined the group upon completion of his medical studies in 1925. The Menninger Foundation was incorporated in 1941.

Dr. Menninger, who had been an honorary member of the Shawnee County Medical Society since 1940, was an active member for many years and at one time served as president of the group. In addition, he held membership in the American College of Physicians, the American Psychiatric Association, and the Central Neuropsychiatric Association.

ACTIVITIES OF MEMBERS

Dr. Glenn R. Peters, president of the Wyandotte County Medical Society, was guest speaker at a meeting of the Personnel Managers' Club of the Kansas City Chamber of Commerce at the Town House Hotel on November 20. His subject was industrial medicine.

Dr. Roscoe T. Nichols, Hiawatha, was honored by the Masonic Lodge in that city on November 17 in recognition of 50 years of membership.

Physicians of Emporia were guests of St. Mary's Hospital, Emporia, on the occasion of the hospital's 25th anniversary. At a program which followed the dinner celebration, Dr. Thomas P. Butcher presided. Other speakers were Dr. F. A. Eckdall, Dr. Clyde Wilson, and Dr. J. J. Hovorka.

Dr. A. E. Hiebert and Dr. H. W. Brooks, Wichita, were speakers before a dental convention held in Wichita last month. Both spoke on "Cancer of the Mouth."

Dr. H. J. Stacey, who has practiced in Leavenworth for 57 years, announced his retirement recently. He and Mrs. Stacey have moved to Oklahoma City to make their home with a daughter.

Dr. Franklin D. Murphy, chancellor of the University of Kansas, was one of the speakers at a conference of presidents and presidents-elect of state medical auxiliaries in Chicago last month.

Dr. Glen C. Hutchison, Hays, and Dr. Lorin E. Dickelmann, Hutchinson, were speakers at a meeting of the Kansas Medical Technologists' Association at Hays last month. Dr. Hutchison spoke on "Laboratory Analysis in Diabetes," and Dr. Dickelmann discussed "Clinical Aspects of Flame Spectrophotometry."

Dr. W. G. Norman and Dr. Fred Gasser, veteran Cherryvale physicians who have practiced there 45 years and 34 years, respectively, were honored at a community dinner in Cherryvale on November 19. Sponsor of the event was the local Chamber of Commerce. A physician new in Cherryvale, Dr. Lee Kramer, was introduced. Speaker of the evening was Dr. R. G. Carter, Independence, president of the Montgomery County Medical Society.

Dr. D. S. Roccaforte, Hanover physician now stationed with the Air Force at Smoky Hill Air Base, Salina, was recently promoted to the rank of captain.

Dr. F. H. Buckmaster, Elkhart, described emotional illnesses to the Lions Club at Elkhart at a meeting held last month.

Dr. Ward E. Benkleman, who has been practicing in St. Francis during the past two years, has moved to Folson, Montana, and will soon open an office there.

Taking part in a Cancer Nursing Institute held at St. Francis Hospital, Wichita, early in December were the following Wichita physicians: Dr. Christine Thelen, Dr. James H. Holt, Dr. Hoyt C. Blaylock, Dr. Dale W. Peters, Dr. A. E. Hiebert, Dr. Jack W. Graves, Dr. Edward X. Crowley, Dr. John R. Kline, and Dr. W. G. Cauble.

Among the speakers at a meeting on lung cancer held at the sanatorium at Norton on November 22 were Dr. Martin J. FitzPatrick and Dr. Galen M. Tice, University of Kansas Medical Center; Dr. John L. Lattimore, Topeka pathologist, and Dr. George W. Jackson, Topeka, director of state institutions, State Department of Social Welfare.

Dr. Stephen John Kelly, Wichita, has been discharged from the U. S. Air Force and has begun a residency at the Jefferson-Hillman Hospital at Birmingham, Alabama.

Dr. Raymond A. Schwegler, assistant clinical professor of gynecology and obstetrics at the University of Kansas School of Medicine, and Dr. Karl A. Menninger, Topeka, were among the speakers at a psychosomatic institute in Topeka in December. The program was devoted to a study of the relation of emotional factors to the female reproductive system.

An advisory committee of two doctors to represent each of Kansas City's hospitals in a United Hospitals Building Fund Campaign was appointed recently as

follows: Dr. Albert C. Harms and Dr. Leland Speer, Bethany Hospital; Dr. Victor B. Buhler and Dr. H. H. Hesser, Providence Hospital; Dr. D. N. Medearis and Dr. F. J. Strick, St. Margaret's Hospital, and Dr. William A. Love and Dr. C. W. Alexander, Douglass Hospital.

Dr. William L. Valk, Dr. Ferdinand C. Helwig and Dr. Albert T. Steegman, all of Kansas City, took part in the scientific program at the A.M.A. meeting in St. Louis last month. Among the scientific exhibitors were Dr. V. E. Chesky, Dr. W. C. Dreese and Dr. C. A. Hellwig, Halstead, and Dr. E. Grey Dimond, Kansas City.

Topekans who took part in the program for an institute on cancer nursing held at Stormont-Vail Hospital, Topeka, early in December were Dr. Robert H. Riedel, Dr. A. A. Fink, Dr. C. Henry Murphy, Dr. D. B. Foster, Dr. Robert M. Brooker, Dr. Orville R. Clark, Dr. H. L. Hiebert, and Dr. Willis L. Beller.

Dr. Orville R. Clark, Topeka, presented a paper, "Traumatic Chylothorax," at a meeting of the Western Surgical Association held in Chicago early in December.

Dr. C. J. Kurth, Wichita, took part in a panel discussion on "The Mental Health Problem in Industry" at a meeting of the Kansas Association for Mental Health in Wichita early last month.

Dr. George W. Jackson, Topeka, director of state institutions, has been named to a new committee established by the American Psychiatric Association to certify physicians as qualified mental hospital administrators.

Dr. LeRoy A. Calkins, chairman of the Department of Gynecology and Obstetrics at the University of Kansas Medical Center, presented a refresher course at the Minnesota University School of Medicine on December 3 and 4. He also addressed the Minnesota Obstetrical Society on December 5 and the Chicago Gynecological Society on December 6.

Dr. Charles F. Haughey has completed a three months postgraduate course in surgery and dermatology at the Cook County Graduate School of Medicine, Chicago. He plans to spend January and February in New Orleans for additional courses, and has announced that he will return to his practice in Tribune in March.

Dr. Robert L. Newman, of the University of Kansas Medical Center, recently became a fellow of the American College of Surgeons. Early in December he attended a meeting of the American Academy of Gynecology and Obstetrics in Cincinnati and there conducted a round table discussion on "Cancer of the Ovary."

Dr. Warren F. Bernstorf, Winfield, was recently elected president of the Kansas Association of School Boards.

Dr. William H. Crouch, Jr., Topeka, has completed the examinations of the American Board of Pediatrics and is now a diplomate of the board.

Dr. E. C. Beaty, Parsons, who recently returned after a tour of duty with the Navy, has been appointed assistant division surgeon of the Katy Railroad, to serve at the Katy Employes Hospital, Parsons.

Three Wichita physicians, Dr. Ernest W. Crow, Dr. Philip W. Russell, and Dr. LeRoy Williams, recently became diplomates of the American Board of Internal Medicine.

Dr. Chester M. Lessenden, who was recently released from active duty with the Navy, is now practicing dermatology in Topeka in association with Dr. Ernest H. Decker. Dr. Lessenden took his specialty training in New York City and is a diplomate of the American Board of Dermatology.

At the regular staff meeting of the Community Hospital, Beloit, new officers for 1954 were elected as follows: Dr. H. L. Collins, president; Dr. R. E. Bennett, vice-president, and Dr. R. P. Weltmer, secretary-treasurer.

Alumni Group Elects Officers

Officers of the University of Kansas Medical Alumni Association, elected at a meeting held recently in Kansas City, are as follows: president, Dr. Kenneth Cox, Kansas City; first vice-president, Dr. Alfred J. Horejsi, Ellsworth; second vice-president, Dr. Mahlon H. Delp, Kansas City; secretary-treasurer, Dr. Wray Enders, Kansas City.

COUNTY SOCIETIES

A meeting of the Ford County Medical Society was held last month at the Lora-Locke Hotel, Dodge City, with Dr. M. J. Cox, president, presiding. Speaker of the evening was Dr. Paul H. Wedin, Wichita, who discussed "Surgical Conditions of the Chest."

A scientific meeting of the Sedgwick County Society was held at the Lassen Hotel, Wichita, on December 1. Dr. Sloan Wilson, of the University of Kansas School of Medicine, spoke on "Recognition and Treatment of False Anemias and Polycythemias."

In observance of "diabetes week," the McPherson County Society sponsored a case-finding drive in that county. Free check-ups for diabetes were offered by all members of the society during the week.

The monthly meeting of the Bourbon County Society was held in November at the Burke Street Hospital, Fort Scott. Dr. Henry Aldis was in charge of the program.

A meeting of the Johnson County Society was held at the Hotel Olathe on November 24. The program for the evening was a scientific film on the treatment of peptic ulcers.

Dr. B. C. Gradinger, Halstead, presented the scientific portion of the program at a meeting of the Harvey County Society held on December 7. The subject of his paper was "Unusual Tumors of the Colon."

Dr. Francis T. Collins was installed as president of the Shawnee County Society at a meeting held at the Hotel Jayhawk, Topeka, on December 7. The following officers were elected to serve with him during 1954: president-elect, Dr. D. Bernard Foster; vice-president, Dr. H. W. Powers; secretary, Dr. Willis L. Beller; treasurer, Dr. David E. Gray; member of board of directors, Dr. Dwight Lawson; member of board of censors, Dr. John A. Segerson; members of the medical service board, Dr. Floyd C. Beelman and Dr. Newman V. Treger.

Delegates to the Kansas Medical Society meeting will be: Doctors Francis T. Collins, John Crary, W. H. Crouch, R. Dale Dickson, A. A. Fink, G. Bernard Joyce, Howard U. Kennedy, W. O. Martin, and Walter Mau. The following will be alternates: Doctors Horace T. Greene, Richard Greer, John Kennedy, Donald H. Macrae, Donald Pierce, Ralph Preston, L. L. Saylor, Daniel L. Tappen, and D. J. Weber.

The program for the evening consisted of a dinner, at which the doctors' wives were guests, and a humorous program with Mr. Martin Hughes, Chicago, as speaker.

About 130 members of the Southeast Kansas Medical Society and their wives attended a dinner meeting held at the Hotel Besse, Pittsburg, December 9. Dr. W. Clarke Wescoe, dean of the University of Kansas School of Medicine, was speaker of the evening, and Dr. Lucien R. Pyle, Topeka, president of the Kansas Medical Society, spoke briefly.

Dr. J. Warren Manley was elected president of Wyandotte County Society at a meeting held at the Town House Hotel, Kansas City, on December 15. The following officers were chosen to serve with him: Dr. L. E. Leigh, vice-president; Dr. Lee H. Leger, secretary; Dr. Alfred Steinzeig, treasurer; Dr. P. E. Hiebert, to the board of censors, and Dr. William F. Roth, Jr., Dr. S. R. Friesen, Dr. M. R. FitzPatrick, and Dr. C. Omer West to the group of official delegates.

A talk, "Problems Facing the Kansas Medical Society," was given by Dr. Lucien R. Pyle, Topeka. Dr. H. Penfield Jones, Lawrence, councilor for that district of the state, also spoke.

A meeting of the Montgomery County Society was held at the Booth Hotel, Independence, December 16. Guests at the meeting were Dr. Lucien R. Pyle and Mr. Oliver E. Ebel, Topeka, and the wives of members of the county society. Dr. Pyle was the speaker of the evening.

Officers for 1954 were chosen by the Barton County Society at a meeting held last month at the Gateway Country Club, Hoisington. The following were elected: president, Dr. Marvin O. Steffen; vice-president, Dr. L. J. Robison; secretary-treasurer, Dr. Clair H. Cavanaugh; delegate to state meeting, Dr. Lucien R. McGill, and alternate, Dr. Robert C. Polson.

Dr. Adolph R. Mueller was elected president of the Leavenworth County Society at a meeting held at the Hotel Cody, Leavenworth, December 14. Dr.

Kenneth L. Graham was named vice-president, Dr. Tom W. Graham was re-elected secretary-treasurer, and Dr. G. Ralph Combs was chosen to serve on the board of censors with Dr. Melvin Rabe and Dr. Mueller.

Dr. Robert E. Bolinger, of the University of Kansas School of Medicine, presented the scientific program on protein diet therapy. At the business session, the group approved a plan for a change in the councilor district to which it belongs.

Facilitating Nurse Recruitment

A new tool for those engaged in student nurse recruitment efforts is now available, it was announced recently by the Committee on Careers, National League for Nursing. A 68-page booklet in looseleaf form, *Manual for Student Nurse Recruiters,* was printed through a grant from the National Foundation for Infantile Paralysis.

The manual covers a wide scope of subjects and gives case histories of successful student nurse recruitment efforts. To help meet the health needs of the nation, the flow of new students into nursing must augment the 102,019 students currently enrolled in 1,148 professional schools and the additional 6,500 students now enrolled in practical nursing schools.

Included in the book are sections on how to organize a student nurse recruitment committee on local or state level, importance of community representation on the committee, and a definition of duties for each of the volunteer workers. Among the programs listed are speakers' bureaus, liaison work with school counselors, school librarians, women's clubs, church groups, parent-teacher associations, and civic, service, and veterans' organizations. Special emphasis is placed on scholarships for student nurses who might otherwise be unable to enter nursing.

Pictures of Polio Virus

Authenticated pictures of an isolated poliomyelitis virus were shown for the first time in November before a meeting of the Electron Microscope Society of America. The presentation was made by Dr. A. R. Taylor, research virologist at Parke, Davis and Company, Detroit, in a paper entitled "Electron Microscopy of Poliomyelitis Virus."

The pictures, some magnified 200,000 to 300,000 times, showed the poliomyelitis virus to be a small spherical particle about 30 millimicrons in diameter. One millimicron is equal to 1/25,000,000 of an inch. The unique pictures thus confirmed previous estimates that the polio virus is one of the smallest of all viruses.

Dr. Taylor explained that the Parke-Davis work was done in connection with the development of a polio vaccine and "indicates the feasibility of developing concentrated virus preparations necessary for the production of a vaccine." The company reports that a vaccine will not be ready for general use until after further development and extensive clinical tests, which may require a year or more.

Geriatric Research

A program of research focused on a group of diseases primarily characteristic of the aged has been begun by the William S. Merrell Company, Cincinnati. Studies are being conducted on atherosclerosis, heart disease, high blood pressure, degenerative diseases, mental illnesses, and functioning of the brain. Expansion of research in endocrinology is continuing.

Merrell's gerontological laboratory is the fourth major expansion project undertaken since 1946.

Postdoctoral Fellowships Available

The National Research Council has announced that Eli Lilly and Company is offering postdoctoral fellowships in the medical and natural sciences to physicians who are under 36 years of age. Applicants must agree to devote themselves to research in anatomy, biochemistry, biophysics, histology, immunology, microbiology, pathology, pharmacology, physiology, and related subjects.

Application blanks may be obtained from the Fellowship Office, National Research Council, 2101 Constitution Avenue, N.W., Washington 25, D. C.

Fellowships in Industrial Medicine

Eight fellowships in industrial medicine will be offered by the U. S. Atomic Energy Commission for the 1954-1955 academic year. The fellowship program, begun four years ago, is designed to provide advance training and experience for physicians in the field of industrial medicine.

The program is administered for the AEC by the Atomic Energy Project of the School of Medicine and Dentistry, University of Rochester, Rochester, New York. Applications should be submitted by January 1, 1954, to Dr. Henry A. Blair, AEC Fellowships in Industrial Medicine, at the Rochester address.

Annual meeting, Kansas Medical Society, May 2-6, 1954, Topeka.

THE MONTH IN WASHINGTON

Editor's Note. The following summary of Washington news was prepared by the Washington office of the A.M.A. for distribution to state and regional medical journals.

The second session of the 83rd Congress is getting down to its task under conditions that could mean passage of considerable legislation of importance to medicine. Holding over from last session, or certain to be introduced this year, are bills touching on virtually every phase of medicine where the federal government could become involved. New laws are being proposed on veterans' care, social security, national health plans, care of military dependents, medical scholarships for military personnel, and many other subjects.

What will be done with this mass of legislation depends on an administration whose control over Congress is tenuous and a Congress looking forward to the fall, when all members of the House and one-third of the Senate must be elected or reelected. As is the case every two years, most lawmakers will be listening closely to what's being said back home.

Awaiting congressional action is the administration's plan for extending the social security system to bring more than 10,000,000 additional persons, including physicians, under Old Age and Survivors Insurance (OASI). This legislation is known to have less support in the House Ways and Means Committee, where it is being handled, than it has in the Executive Branch.

American Medical Association, supported by dentists, lawyers, farmers, and many other groups of self-employed, has consistently opposed inclusion under OASI. The question now is whether this opposition will be articulate enough to convince Congress.

In place of social security for physicians, the A.M.A. for several years has actively promoted legislation identified first as Reed-Keogh, then as Jenkins-Keogh, named for the sponsoring congressmen. This would allow physicians and other self-employed to defer income tax payments on a portion of their income, placed in restricted pension funds, obtainable in the form of benefits only in case of disability or at the specified retirement age. In this effort the physicians again are joined by a large group of associations representing the self-employed.

Other possible amendments to the social security law involve total and permanent disability payments and waiver of OASI premiums for the disabled, so their final pensions won't be reduced because of periods when they had little or no income. In each of these, medical determinations would be required. In the past, these bills have threatened an expansion of the federal medical program, have laid out an unreasonable role for the physician, or have called for compulsory rehabilitation. While not opposed to the objectives, A.M.A. has urged that both the patient and the physician be protected. In place of waiver of premium, the A.M.A. proposes that pension rates be based on the 10 best earning years, thus obviating the need for medical determinations.

As in other sessions, Congress this year probably will be asked to pass legislation providing free hospitalization under OASI for all persons past 65 covered by OASI, and for other beneficiaries of the program. In other years Congress has not taken this idea seriously.

The veterans program is certain to provoke action. Last November, Veterans Administration amended its forms to require more financial information from veterans applying for hospitalization of non-service connected disabilities, who must state that they cannot afford private care. Congress may want to further clarify the government's obligation to veterans. It is expected also that special effort will be made to expand medical benefits for veterans by such methods as increasing the periods in which certain diseases may be presumed to be of service origin.

The A.M.A.'s position on the care of non-service connected cases is well known. It consists of three points. First, the best possible care by VA for actual service-connected cases. Second—until local and state facilities are adequate—VA care for long-term tuberculosis and neurological cases when the veteran himself can't pay. Third, all other non-service connected cases to be the responsibility of the veteran himself, his family, or his community.

The Defense Department has served notice that this session it will press hard for implementation of the Moulton Commission's recommendations for broadening the medical care program for military dependents. The Commission favored caring for as many dependents as possible at military installations, with the others receiving private care and the federal government paying all but a token of the cost. At its December meeting, the A.M.A.'s House of Delegates proposed that in this country the military provide medical care for dependents only where private facilities are not adequate.

Also up for decision this year is a Defense Department's proposal that the federal government furnish medical, dental and nursing scholarships, with the recipients obligated for government service at the rate of one year for every year of the scholarship.

There is a strong possibility of pressure to enact a program under which the federal government would

in one way or another subsidize private health insurance plans. The idea is known to interest Rep. Charles Wolverton (R., N.J.), chairman of the House Interstate and Foreign Commerce Committee, which last fall conducted a series of hearings on health matters. Senators Ives (R., N.Y.) and Flanders (R., Vt.) are offering a bill along the same lines in the Senate.

The controversial Bricker resolution holds over from the last session, and may receive early consideration in the Senate. Senator Bricker believes that Congress should have some check on the President's treaty-making powers. The American Medical Association repeatedly has indorsed the Bricker resolution as a safeguard against the introduction into this country by treaty of government-controlled medical plans without Congress itself having a chance to pass on them.

Awaited with interest in Washington are the findings of two Commissions appointed last year to look into the relationships between the federal government on the one hand and state and local governments on the other, and to investigate operations of the executive branch. The former is headed by Clarence Manion and the latter by former President Hoover. The Hoover Commission has until next year to make its report. The Manion Commission was instructed to have a report ready by March, but it may ask for more time.

ANNOUNCEMENTS

The Ninth National Conference on Rural Health, sponsored by the A.M.A.'s Council on Rural Health, will be held in the Baker Hotel, Dallas, March 4-6. The conference will stress the importance of individual participation in community affairs, and the theme will be "Let's Put More 'U' in CommUNITY."

The formal sessions will begin at 2:00 p.m. on March 4, preceded by a pre-conference meeting "for doctors only." That session will be concerned with two items, physician participation at the grass roots and health insurance matters.

The National Multiple Sclerosis Society and the National Institute of Neurological Diseases and Blindness are distributing a new pamphlet designed for consumption primarily by the victims of multiple sclerosis. It is a realistic presentation of the medical status of the disorder and the facilities available for its amelioration.

Physicians who see patients who will find the booklet helpful are asked to advise such patients that the booklet is available from the Public Inquiries Branch, Public Health Service, Department of Health, Education, and Welfare, Washington 25, D. C.

The Third Interim Congress of the Pan American Association of Ophthalmology will be held in Sao Paulo, Brazil, June 17-21, 1954. Meeting concurrently in Sao Paulo will be the Eighth Brazilian Congress of Ophthalmology and the International Congress of Oto-Neuro-Ophthalmology. Translations of all proceedings will be offered in three languages, including English. Attendance is not restricted to members. The registration fee is $10.

The Third International Congress of Internal Medicine will be held in Stockholm, September 15-18, 1954, under the auspices of the International Society of Internal Medicine in which 40 nations are represented.

The 18th annual midwinter postgraduate clinics sponsored by the Colorado State Medical Society for the Rocky Mountain region will be held at Denver, February 16-19, 1954, with headquarters at the Shirley-Savoy Hotel. The program, designed especially to interest general practitioners, will consist of three mornings of "dry" clinics, three noon round-table luncheon discussions, and three afternoons of illustrated lectures by guest speakers. A dinner dance will be held on the evening of February 18.

Registration is open to all doctors of medicine and their guests. A fee of $5.00 will be charged, but students, interns, full-time residents, doctors' families and other non-physicians are exempt.

The International Academy of Proctology announces its annual cash prize and certificate of merit award contest for 1953-1954. Awards will be given for essays on proctology and allied subjects, not exceeding 5,000 words in length. Entries must be received no later than February 1, 1954. The address is International Academy of Proctology, 43-55 Kissena Boulevard, Flushing, New York.

A sectional meeting of the American College of Surgeons will be held March 1-4, 1954, at Omaha, Nebraska, with headquarters at the Hotel Fontenelle. Each morning there will be clinics at Omaha hospitals, and each afternoon will be devoted to scientific programs. The faculties of the medical schools at the University of Nebraska and Creighton University are co-operating in the presentations.

Because of the popularity of this type of program, an invitation to attend is extended to all surgeons, without regard for fellowship in the College. Hotel reservations should be made immediately.

BLUE SHIELD

New Enrollment Experiment

A new Blue Cross-Blue Shield community enrollment program for 1954 has been approved by the Boards of Trustees, and as a result annual enrollment dates will be established in every county.

In other words, Kansans will now know ahead of time when Blue Cross-Blue Shield will be available in their area. These dates will be the same each year in every county.

Employee group enrollment will continue to be available at any time. Groups may be formed in companies having five or more employees by meeting regular quota requirements.

The new method of community enrollment is made possible by the introduction of a new application form which contains a fairly detailed and comprehensive statement of health. The gist of the health statement is as follows:

1. Has anyone to be covered by your application for membership had or does he now have: cancer, diabetes, asthma, epilepsy, ulcers, high blood pressure, kidney diseases, rheumatic fever, thyroid trouble, physical defects (congenital or otherwise), heart trouble, arthritis, tuberculosis, paralysis? If "yes," please give details (including date of last consultation or treatment, attending physician and address, name of hospital if hospitalized).

2. Has anyone to be covered by your application for membership been hospitalized within the last three years for any condition other than those listed above? If "yes," please answer fully (full name of person, relationship to applicant, condition or ailment, name and address of hospital, name and address of doctor).

3. Give names of regular family physicians.

4. Do you consider all persons to be covered by this application to be in good health and free of disease? If "no," please explain.

5. I authorize any physician or hospital to furnish Kansas Blue Cross-Blue Shield information which may be pertinent to the medical history of all persons to be covered by this application.

6. I have read paragraphs one through three, and to the best of my knowledge, the above information supplied by me is correct and complete.

I further understand that acceptance of this application will be based upon the information contained in it and that any misrepresentation or failure to include information concerning the persons for whom application is made will give Kansas Blue Cross-Blue Shield the right to deny benefits for any conditions misrepresented or not reported on this application.

The purpose of this health statement is to improve the selection of risk among new members. In the past, Blue Cross-Blue Shield has been wide open to those who were in poor health and were perhaps the first to apply for membership in every community enrollment. The health statement will tend to prevent this anti-selection and will put emphasis on people joining before they become poor health risks.

If an applicant checks that he has one of the conditions listed on the application, one of two decisions will be made by the Underwriting Section of Blue Cross-Blue Shield:

1. Whether to accept the membership at all.

2. Whether to accept the membership by attaching a rider to the contract excluding coverage for any medical or hospital care for the condition checked.

It is believed that this manner of handling applications will cause the least misunderstanding and poor public relations later on. The member will definitely know that his contract is not good for the condition specified in the rider. He will know this before he has paid his dues. Other members of the family are not affected by the rider.

In spite of every effort which will be made to have a clear understanding with new members, the utmost co-operation by physicians will be necessary to inform people of the spirit and intent in this new program. Some people are going to resent the health statement. Perhaps some doctors will not like to give information which may prevent their patients from receiving benefits. But the health statement is necessary if a more open enrollment plan is to be followed. The only safe alternative to a strict health statement is group enrollment on a quota basis. Of course, this would be virtually impossible in the non-group category.

This new enrollment program is both more liberal and more strict. It is more liberal in the sense that it will open up enrollment to every county in the state (except Wyandotte and Johnson) at least once a year. It is more strict in that the health statement is being required which will tend to screen out poor health risks.

The understanding of the medical profession is earnestly sought so that the program may be successful.

Attention is called to an error in last month's Blue Shield column. Income limits for service benefits have not been changed. They are $2,000 for the single member and $3,000 for the family member.

Twenty-seven million people are now enrolled in Blue Shield medical care plans, according to an announcement by the Blue Shield Commission in Chicago.

Radioactive Iodine in Hyperthyroidism

Lowell Rhodes, M.D.

Wichita, Kansas

The etiology of hyperthyroidism is still unknown, and, therefore, it is understandable why its treatment remains empirical. The present methods of treatment consist of interruption of the production of thyroxin by antithyroid drugs, destruction or removal of all or part of the thyriod gland by surgery, or radiation of the gland. We are awaiting some drug or some method that will attack the disease at its source and thus make the present methods of treatment obsolete.

It might be said that the development of radioactive iodine, for use in the treatment of diseases of the thyroid gland, really began in 1915-1916, when David Marine[17] definitely established the affinity of the thyroid gland for iodine. The first studies[21] with radioactive iodine itself were done in 1934 by Fermi,[36] who developed it by exposing iodine to tubes containing radium and beryllium powder for 30 minutes. Radioactive iodine, itself, did not become readily available until 1936, following the development of the cyclotron. Fourteen radioactive isotopes have been identified, of which four have been used in biological investigation:[21] I^{126} with a half-life of 13 days; I^{128} with a half-life of 25 minutes; I^{130} with a half-life of 12.6 hours; and I^{131} with a half-life of 8 days.

Among the first institutions to start investigations in 1937 were the Massachusetts Institute of Technology and the Massachusetts General Hospital. They were soon followed by the University of California and others. These studies have done much to solve the problems of the metabolism of iodine and the synthesis of thyroxine as well as the mode of action of some antithyroid drugs. Among the early reports of the use of radioactive iodine in hyperthyroidism were those of Hamilton and Lawrence,[10] and Hertz and Roberts,[13] using a mixture of I^{130} and I^{131}. Hamilton and Lawrence used large doses of I^{131} and reported the complete destruction of the thyroid without damage to other organs in rabbits and dogs. Later they gave smaller doses to human beings, with a resultant drop in the basal metabolic rate.

Miller et al.[18] have stated certain conditions which must be fulfilled in order for any radioisotope to be usable for the therapy of disease. First, the disease process must be capable of being treated successfully by a radioactive substance. The isotope being used must have a physical half-life long enough to be useful, but not so long as to make its use dangerous. It must be concentrated in the tissue being treated, but not in any tissue in dangerous quantities. It must remain in the tissues long enough to permit effective radiation. Its main radiation must be from alpha or beta particles, so that its radiation effects are largely limited to the tissue treated. Small portions of gamma rays should be present, so its activity may be measured outside of the body.

Because of its convenient half-life, and the relatively pure form in which it can now be obtained from the uranium chain reacting pile, I^{131} with a negligible contamination of I^{133} is now almost universally used in both investigation and therapy.[31] With quantities of radioiodine, as discussed later, neither objective nor subjective evidence of injury to the kidneys, liver, salivary glands, or stomach has been reported,[18] and it is to be considered safe in the light of our present knowledge.[4, 32] Even overdoses, up to 100 times the therapeutic dose, do not seem to damage other organs.[6] The possibility of late radiation damage leading to malignant change cannot be ignored; however, a concensus of opinion based on experience with x-ray treatment of toxic goiter indicates that late malignant complications with radioiodine therapy are probably unlikely.[32]

Radioactive iodine is given orally. Absorption begins immediately, is about 80 per cent complete in one hour, and is usually complete in two hours.[15] If food is present in the stomach, absorption is delayed. A curve of radioactivity in the blood (radioactive protein bound iodine) has been plotted by Keating,[15] and is seen to have three components: (a) A rapid rise, followed by a brief, rapid fall. This phase represents absorption and the establishment of equilibrium between the blood and other body fluids; (b) A more prolonged fall, the rate depending upon the state of the thyroid and upon renal function. In this phase, if the function of the kidneys is altered, then the renal collection rate is lowered and the fall in the blood is more prolonged;[16] and (c) A flattened out phase, with an occasional rise in hyperthyroid patients. The peak in this curve is reached in myxedematous and normal subjects in two hours, while the peak in those with hyperthyroidism may not be reached for eight hours.[15]

Following absorption, the concentration of the isotope is 10,000 times greater in the thyroid tissue than in any other tissue, according to Crile.[6] From 85 to 90 per cent of the total amount given in a

---

This is one of 11 senior theses selected for publication by the Editorial Board from a group of 15 judged the best by the faculty at the University of Kansas School of Medicine.

Thesis written while the author was a senior student. Dr. Rhodes is now interning at Wesley Hospital, Wichita, Kansas.

tracer dose can be accounted for in the thyroid and excretion in the urine,[16, 32] the urine being the principal route for its disposal in euthyroid and hypothyroid subjects.[16] Sixty-five per cent is excreted in the urine in two days. Almost complete excretion occurs in hypothyroid patients in 4 to 5 days, while normal or hyperthyroid individuals may require several days to weeks for complete excretion.[16] If the urine is collected and tested, a reciprocal relationship is noted between the rate of uptake and the amount excreted.[33] Despite the large amounts of radioiodine that pass through the kidneys, no adverse effects have been noted upon them.[18]

Small amounts are also lost in the feces (1 to 2 per cent) and in sweat. About 10 to 15 per cent is distributed to the various organs and tissues of the body other than the thyroid.[16] By the end of one month, most of the radioactivity is gone, and by two or three months, practically none remains.[6]

The metabolism of radioactive iodine can be dramatically influenced by a variety of things. Thyroid extract given before administration causes a depression in the rate of uptake. Werner[33] noted that as little as .12 grams permitted an uptake of only 4 per cent. He and others[22] have shown that iodine lowers the uptake. The normal values of uptake for adults are not necessarily those found in children,[24] who may show a low uptake but are not clinically suspected of being hypothyroid. And then in normal aging, there is a decrease in the basal metabolic rate which may be correlated with atrophy of the acini, decrease in colloid and iodine content, and a decrease in the number of dividing cells.[22] Sex plays a part, too. The rate of uptake of young males and females may be about the same, but the mean gradient of the females is higher than the corresponding mean of the males in both the young adult and the older age groups.[22]

The rate of uptake also varies in certain hormone disturbances. Werner[33] reports a lowered uptake in panhypopituitarism and in adrenal cortical hyperfunctioning, while Reiss[26] reports a decrease in adrenal cortical insufficiency. The uptake is reported by Reiss[26] to be increased when given simultaneously with .3 mg of epinephrine. Sofer, Gabrilone, and Jailer report a decreased uptake of I^{131} when given with epinephrine in intact rats, but the uptake is markedly increased if I^{131} is given simultaneously with epinephrine in adrenalectomized rats.[30]

Dyes containing iodine used in x-ray studies will influence the uptake for varying periods of time. Those used in gallbladder visualization and bronchography will affect uptake for several months, while the effects of those used in intravenous pyelograms last several weeks.[31] Most of these factors which cause variations can be elicited on questioning or examination and should not be of great moment to the clinician when they are known.

In recapitulation, the use of radioactive iodine in hyperthyroidism is justified by its collection by the thyroid gland shortly after administration and the rapid excretion by the kidney of that not taken up by the gland. The thyroid gland can be destroyed by I^{131} in doses that show no damaging effect upon any other organ or tissue in the body. And lastly, the clinician must be aware of the conditions which will produce a variation in iodine metabolism.

Having determined that we have an agent for use in thyroid disease, the next question is, how shall we use it in thyroid evaluation? There are three methods of study open to us. First, we may measure the percentage uptake of radioactive iodine by the thyroid during a given period of time under standardized conditions. This will be discussed later. Second, we may measure the urinary excretion of the isotopes in a given period of time under controlled conditions. This method will not be considered here, due to inaccuracies which are attributed to improper collection of the specimen, failure to collect a complete specimen, and the more technical task of determining the radioactivity.[16, 33] The third method is that of radioautographic prints of tissue. This method, though of little use to the clinician in his office practice, will be discussed because of its value in helping us to understand some of the problems of thyroid disease.

Dobyns and Lennon[7] and Dobyns, Skane, and Maloof[8] have noted a difference in the state of functioning of thyroid adenomas with radioautographs of these tumors. Their method consists of giving patients I^{131} 24 to 72 hours before surgery, removing nodules, making blocks, cutting and mounting tissue, and then exposing the tissue to film for varying lengths of time to obtain prints. They make the assumption that the degree of affinity for radioactive iodine represents the degree of function of the thyroid tissue. Then, by staining and studying the slides, and comparing the slides with the radioautographs, they have noted several types of adenomas.

There are adenomas which have cellular hypertrophy and hyperplasia and which hyperfunction, and there are also adenomas which have cellular hypertrophy and hyperplasia and which scarcely function at all. The group with little function shows an increased variability in cell height and appears to shade off into carcinoma.[7] Dobyns, Skane, and Maloof[8] constructed a Geiger counter suitable to count small areas of a gland, permitting them to determine the activity of a nodule. Then it may be concluded[6] that if a single nodule is found to have distinctly less function than the extra-nodular tissue, this will exclude the hyperfunctioning group and increase the suspicion of malignancy. Without such a special Geiger counter, the clinician cannot tell the activity of the nodule and will have to rely completely on his clinical judgment in therapy.

The usual method of determining the percentage uptake of radioactive iodine by the thyroid gland is initially a determination of the activity of 40-100 microcuries of I^{131} with a Geiger counter. The agent is then given orally to the patient in water, in a paper cup. The cup is rinsed with water, and the patient drinks the rinse water. The patient returns in 24 hours, at which time the activity is determined over the thyroid gland, again by means of a Geiger counter,[11] and the percentage uptake is computed. Werner, Quimby, and Schmidt[32] found the normal uptake in 24 hours to lie between 10 and 40 to 50 per cent, with a mode between 15 and 30 per cent. In nearly all cases of hyperthyroidism, more than 40 per cent of the administered dose was found within the thyroid at the end of 24 hours; in subjects with myxedema, the corresponding figure was usually less than 10 per cent.[33] If the urine excretion should be calculated, it is found that euthyroid subjects excrete from 52 to 84 per cent of the administered dose with an average of 66 per cent in the urine.[15] For thyrotoxic subjects, the range of excretion was 6 to 32 per cent with an average of 18 per cent. Hypothyroid subjects excrete 35 per cent of the dose.[11] The results should then be correlated with other clinical data.[33]

Having decided that a patient has hyperthyroidism, careful selection of patients to be treated with radioactive iodine should be made, in order to secure satisfactory results. Since therapeutic activity depends upon beta radiation, which penetrates tissue for little more than two millimeters,[4] the major field of usefulness[1, 4, 20] is in the diffusely hyperplastic goiters which cannot be more readily and satisfactorily treated in some other way. The selected patients should be those: who fail to respond to apparently adequate dosage of an antithyroid compound, or who have repeated relapses after such treatment; who are sensitive to antithyroid compounds; who would be a surgical hazard due to age, heart disease, bleeding dyscrasias, and such; who have a recurrence of toxicity after repeated surgical procedures; or who have had damage to a vocal cord. Because the possibility of causing a malignancy due to radiation cannot be completely discounted, the majority of investigators prefer to use I^{131} only in patients of the older age group, although malignancies due to radiation are usually associated with chronic, low intensity exposure, and not brief radiation.[2] It is generally conceded that surgery is the treatment of choice in single adenomas where carcinoma may be present;[20] however, I^{131} may be an acceptable type of treatment in patients with nodular goiters in the older age groups who are poor surgical risks, or in those who definitely oppose operation.[20]

Contraindications to I^{131} treatment are few. Since the fetal thyroid is capable of absorbing and concentrating iodine as early as the 14th week, it seems unwise to give an isotope with a half-life of 8 days to a woman who is beyond the 10th week of pregnancy.[3, 5] It has been felt that renal disease represents a condition in which therapeutic doses of radioiodine might do considerable harm. However, Williams et al.[35] have reported no increase in radioiodine in the kidneys of patients with severe glomerulonephritis.

When it is decided that a patient is to be treated by I^{131}, his uptake is determined and all iodine and thiouracil-like therapy is withheld for two[31] to four days.[4, 20] The therapeutic dose is then given in a manner similar to the tracer dose as described above. The amount to be given depends upon:[1] the fraction concentrated in the thyroid; the weight of the thyroid gland; the disintegration scheme of the isotope; the time necessary for decay of radioactivity to zero, and the rate of elimination from the gland.

Obviously the greatest chance for error lies in the estimation of the size of the gland. Shipley and his associates[29] estimate the size of the gland by assuming the normal gland to weigh 20 grams; the lower limit of an enlarged gland is about 35 grams, and the largest gland weighs about 100 grams. A satisfactory dose for therapy probably lies between 100 and 150 microcuries per estimated gram of tissue in diffuse goiters,[4, 28] although all factors must be taken into account in any given case. For nodular goiters the dose is about 240 microcuries per gram of tissue.[28] There are cases reported where as much as 500 microcuries per estimated gram of tissue have been given without permanent myxedema.[14] Some workers determine the dose by estimating the amount of radiation necessary, and then converting this to microcuries, but since they do not agree on the amount of radiation necessary, estimation of the size of the gland is satisfactory.[23, 28]

Clinical improvement usually appears about the middle of the second week, but remission is not established until the end of the second or third month.[19, 29, 32] During this time thiouracil treatment may be necessary. It may be started in from 5 to 7 days after treatment.[31] Actual shrinkage of the gland may not appear for three months.[29, 32] At the present time it appears possible to obtain therapeutic success in 75 to 90 per cent or more of selected patients with hyperthyroidism.[2, 31]

If after two months there is no improvement or only slight improvement, further treatment may be necessary.[19] If the basal metabolic rate is unchanged, the first dose should be doubled. If it is one-half its original amount, give a dose of I^{131} equal to the original dose, and if it is 2/3 of the way to normal, give about one half of the original dose.[6]

If the response in nodular goiters is not what was expected, it may be because:[25, 27] the I^{131} gets lost

The Problem of Nausea and Vomiting:

ITS TREATMENT WITH DRAMAMINE®

Whenever nausea, vomiting and vertigo are disturbing and complicating factors, Dramamine may be used with confidence.

Keats[1] outlines the wide list of conditions in which Dramamine (brand of dimenhydrinate) has proved valuable as follows: "It has been well established in the control of motion sickness. It has been used effectively in the prevention and treatment of seasickness, airsickness, [in the treatment of] the nausea of pregnancy, Ménière's syndrome, . . . radiation sickness . . . and postfenestration reactions. . . . The site of action is imperfectly understood, but there is indication of an action of depressing labyrinthine function or its neural pathways, a highly selective central action, or both. Few side reactions of this drug have been noted."

The usual dose for motion sickness is 50 mg. (one tablet) taken one-half hour before departure and, if necessary, before meals for the duration of the journey. Control of nausea and vomiting of other conditions and severe motion sickness is achieved, with minimal drowsiness, by a dosage of 100 mg. every four hours.

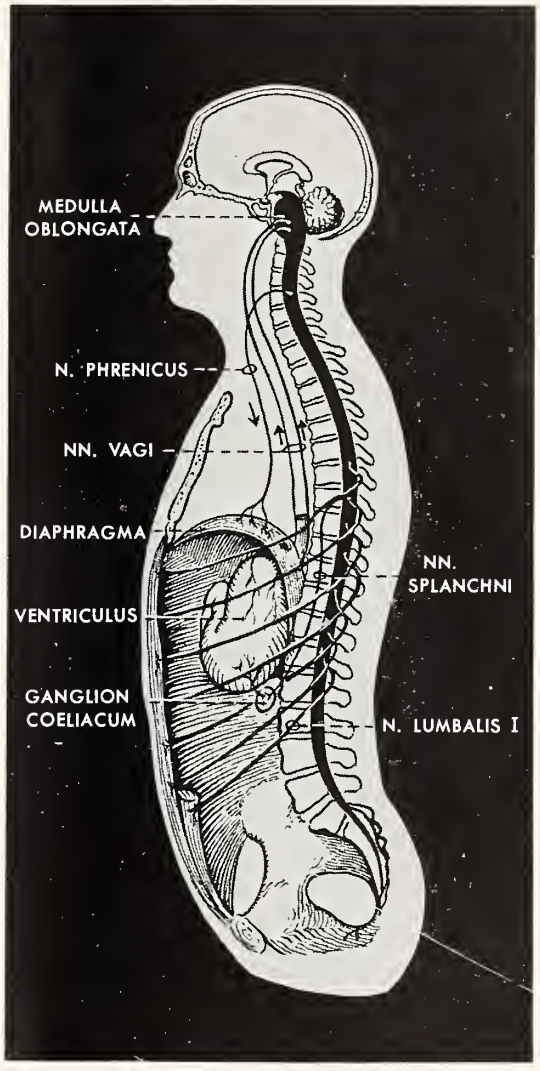
"[Dramamine] is administered orally or rectally. . . . The same doses may be administered rectally by insertion of the tablet or other suitable form. . . ."[2]

Dramamine Liquid is particularly useful for children.

Dramamine is accepted by the Council on Pharmacy and Chemistry of the American Medical Association.

1. Keats, S.: Ataxic Cerebral Palsy with Akinetic Seizures: Dramatic Response to Dramamine, J. M. Soc. New Jersey *50*:53 (Feb.) 1953.

2. Council on Pharmacy and Chemistry: New and Nonofficial Remedies, 1953. Philadelphia, J. B. Lippincott Company, 1953, p. 471.



THE VOMITING REFLEX: *Vagus→ nodose ganglion→ solitary tract→ spinal cord→ cervical, thoracic and lumbar nerves to diaphragm, cardiac sphincter, stomach, abdominal and pelvic musculature. (After Krieg, W. J. S.: Functional Neuroanatomy, ed. 2, New York, The Blakiston Company, Inc., 1953, p. 104.)*

SEARLE *Research in the Service of Medicine*

in the colloid of a huge nodular goiter, and, being relatively far away from cells, fails to irradiate them effectively; the nodules are in different states of activity, and, as the hyperactive nodules regress, other nodules become hyperactive; nodular goiter is so totally different from the diffuse goiter of Grave's disease that the uptake of I^{131} is totally different, and the response to treatment is different.

Angina pectoris has either disappeared or been greatly ameliorated when hyperthyroidism was controlled. Persistent auricular fibrillation following therapy indicated either continuing hyperthyroidism or severe underlying organic heart disease, necessitating other means of heart control.[25]

A number of complications have been observed as a result of the use of I^{131} in the management of thyrotoxicosis. They are discussed briefly in the following paragraphs.

Acute thyroiditis: In some cases (the number varies with the various authors), tenderness of the gland appears within 3 to 4 days after the radioactive material is administered. This usually subsides in about 10 days, but it has been known to persist for several months.[2, 32]

X-ray sickness: Nausea, vomiting, malaise, and fever seen following roentgen ray therapy may occasionally appear in the course of treatment.[31, 32]

Thyroid crisis: It has been thought that this could occur if too little radioactive isotope is used. However, in experimental rats,[9] it has been shown that it is the largest dose of I^{131} which produces the greatest elevation of values for protein bound iodine in the serum, and hence, might be thought to cause exacerbation of the thyrotoxic state. The rise is probably due to release of breakdown products from the gland into the circulation. All investigators have reported a temporary increase in the thyrotoxicosis in some patients.

Exophthalmos: The tendency for malignant exophthalmos to occur is greatest after surgery and least marked following x-ray therapy,[21] while its incidence with I^{131} occupies an intermediate position.

Myxedema: The incidence of this complication has varied with different groups of observers throughout the country and has been transient in many instances. In some cases, desiccated thyroid is given for three months, and then it is no longed needed.[35] Its incidence increased with very large doses of the isotope, it is rarely seen when the thyroid uptake does not exceed 100 microcuries per estimated gram of tissue, but it has been observed when as little as 35 microcuries per gram of tissue were used.[34] Twenty to 25 per cent may become hypothyroid.[2, 31]

Cancer: As has been stated previously, the possibility that cancer may follow the beta radiation with I^{131} cannot be denied, although no cases have thus far been reported in which such an association has been considered likely. Because the possibility cannot yet be completely discounted,[4] the majority of investigators prefer to employ I^{131} only in patients of the older age group, although malignancies due to radiation are usually associated with chronic, low intensity exposure, and not brief radiation.[2]

Some of the advantages of radioiodine over other forms of treatment are: (1) the simplicity of the procedure; (2) lack of interference with the patients' routine of living; (3) absence of skin lesions seen following roentgen ray therapy; (4) no mortality due to this type of treatment;[1] and (5) the relative infrequency of toxic manifestations.[1]

The disadvantages include: (1) the necessity for trained personnel and expensive equipment; (2) special precautions for handling a radioactive material; (3) the possible influence of radioiodine upon the urinary tract;[21] and (4) difficulty in ascertaining the evenness with which the effect has been exerted throughout the thyroid gland.

SUMMARY

1. Radioactive iodine has become a valuable agent in the study, the differential diagnosis, and the treatment of thyroid disease.

2. The development of radioactive iodine treatment is dependent upon the work of Marine, who showed the affinity of the thyroid gland for iodine, Fermi who developed radioactive iodine, and the early work of Hamilton, Lawrence, Hertz, and others.

3. I^{131} has been used in the study of the metabolism of iodine.

4. The rationale of the use of radioactive iodine in thyroid disease is based on physiological experiments and anatomical (or pathological) facts. These are considered.

5. With selection of patients and proper therapy, good results may be expected. The proper selection, treatment and expected results are discussed, as are the contraindications and complications of this type of therapy.

BIBLIOGRAPHY

1. British Med. Journal, Treatment of thyrotoxicosis with radioactive iodine, (Editorial) 2:373 (August 18) 1951.

2. Burrows, B. A., and Ross, J. R.: The management of hyperthyroidism, Med. Clinics of North America, September 1951, W. B. Saunders & Co., Philadelphia and London.

3. Chapman, E. M.; Corner, G. W.; Robinson, David, & Evans, R. D.: The collection of radioactive iodine by the human fetal thyroid, J. Clin. Endocr. 8:717-20 (Sept.) 1948.

4. Clark, D. E.; Trippel, O. H., and Sheline, G. E.: Diagnostic and therapeutic use of radioactive iodine, AMA Archives of Int. Med. 87:17-24 (Jan.) 1951.

5. Clever, J. E.: Radioactive iodine for hyperthyroidism, administered during early pregnancy, Am. J. Ob. and Gyn. 61: 217-219 (Jan.) 1951.

6. Crile, George; McCullagh, E. P., and Glasser, O.: Experience with radioactive iodine in the treatment of hyperthyroidism, Cleveland Clinic Quarterly 16:1 (Jan.) 1949.

7. Dobyns, B. M., and Lennon, B.: A study of the histopathology and physiologic functions of thyroid tumors, using radioactive iodine and radioautography, J. Clin. Endocr. 8:732-748 (Sept.) 1948.

8. Dobyns, B. M.; Skane, B., and Maloof, F.: A method for the pre-operative estimation of function in the thyroid tumors, its

Mutual Fund Investing

Many recent articles in professional magazines have discussed the merit of investing the "Mutual Fund Way" for professional people.

The only sound approach to Mutual Fund Investing is a thorough diagnosis of over 100 available funds, a comparative analysis of performance, policies, objectives, income provided and acquisition costs. The performance and dividends of some funds have been outstanding—others mediocre at best.

Our Mutual Funds Research Department is a virtual laboratory to determine these facts impartially. As investment bankers, we can assist you in determining what fund, if any, we believe best for you.

We suggest, before you *buy* any Fund from any source, you obtain a free report from our Mutual Fund "Laboratory."

TO INVESTIGATE BEFORE YOU INVEST IS BETTER THAN AFTER.

Call or write
For our booklet "Understanding Mutual Funds" or other information

ESTES & COMPANY, INC.

Investment Bankers

112 West 7th Street
Topeka, Kansas **Telephone 2-7231**

significance in diagnosis and treatment, J. Clin. Endocr. 9:1171-1184 (Nov.) 1949.

9. Feller, D. D.; Chaikoff, I. L.; Taurog, A., and Jones, H. B.: The changes induced in iodine metabolism of the rat by internal radiation of its thyroid with I^{131}, Endocrinology 45:464 (Nov.) 1949.

10. Hamilton, J. G., and Lawrence, J. H.: Recent clinical developments in the therapeutic application of radio-phospborus and radioiodine, J. Clin. Invest. 21:624 (May) 1942.

11. Hamilton, J. G., and Soley, M. H.: Studies in iodine metabolism by the use of a new radioactive isotope of iodine, Am. J. Physiol. 127:557-572 (June) 1939.

12. Hamilton, J. G., and Soley, M. H.: Studies of iodine metabolism of the thyroid gland in situ by the use of radioiodine in normal subjects and in patients witb various types of goiter, Am. J. Physiol. 131:135-143 (June) 1940.

13. Hertz, Saul: Radioactive iodine as an indicator in thyroid pbysiology, Am. J. Roentg. & Radium Ther. 46:467 (Oct.) 1941.

14. Johnson, J. B.; Ireland, C.; Thomas, R. F., and Bass, A.: Treatment of hyperthyroidism with radioactive iodine, Am. J. Med. 6:504 (April) 1949.

15. Keating, F. R., Jr., and Albert A.: The metabolism of iodine in man as disclosed with the use of radioactive iodine, Recent Progr. Hormone Researcb 4:429, 1949.

16. Keating, F. R., Jr.; Power, M. H.; Berkson, J., and Haines, S. F.: Tbe urinary excretion of radioiodine in various thyroid states, J. Clin. Invest. 26:1138 (Dec.) 1947.

17. Marine, David: Quantitative studies on the in vitro absorption of iodine by dog's thyroid glands, J. Biol. Chem. 22:547, 1915.

18. Miller, E. E.; Daily, M. E., et al. II. Treatment of patients with hyperthyroidism by I^{131}, Radiology 57:227 (August) 1951.

19. Moe, R. H.; Adams, E. E., et al.: An evaluation of radioactive iodine in the treatment of hyperthyroidism, J. Clin. Endocr. 10:1022-8 (Sept.) 1950.

20. McCullagh, E. P., and Richards, C. E.: Radioactive iodine in the treatment of hyperthyroidism, AMA Archives of Int. Med. 87:6 (Jan.) 1951.

21. McGavack, Thomas H.: The Thyroid, The C. V. Mosby Co., 1951. St. Louis.

22. Perlmutter, M., and Riggs, D. S.: Thyroid collection of radioactive iodide and serum protein bound iodine concentration in senescence, in hypothyroidism, and in hypopituitarism, J. Clin. Endocr. 9:430 (May) 1949.

23. Portmann, V. V.; Hays, R. A.; McCullagh, E. P., and Richards, E. E.: Experience in the treatment of diseases of the thyroid gland with radioactive iodine, Am. J. Roentg. and Radium Ther. 66:179 (Aug.) 1951.

24. Quimby, E. H., and McCune, D. E.: Uptake of radioactive iodine by the normal and disordered gland in children, Radiology 49:201-4 (Aug.) 1947.

25. Radioiodine Therapy in Hyperthyroidism: Cardiac problems, J. Clin. Endocr. 11:1296 (Nov.) 1951.

26. Reiss, R. S.; Forsham, P. H., and Thorn, G. W.: Studies on the interrelationship of adrenal and tbyroid function, J. Clin. Endocr. 9:659 (July) 1949.

27. Richards, C. E.; Crile, Geo., and McCullagh, E. P.: Radioactive iodine in the treatment of hypertbyroidism of nodular goiters, J. Clin. Endocr. 10:1077-1083 (Sept.) 1950.

28. Scott, W. G., and Seaman, W. B.: Observations and results in the treatment of hyperthyroidism with radioactive iodine, I^{131}, Am. J. Roentg. and Radium Ther. 66:171 (Aug.) 1951.

29. Shipley, R. A.; Storansli, J. P.; Freidele, J. L., and Potts, A. M.: I^{131} in the diagnosis and treatment of hypertbyroidism, Am. J. Roentg. 64:576-589 (Oct.) 1950.

30. Sofer, L. J.; Gabrilone, J. L., and Jailer, J. W.: The role of adrenal in the uptake of I^{131} by the thyroid following parenteral administration of epinephrine, Proc. Soc. Exp. Biol. & Med. 71:117 (May) 1949.

31. Sweeney, B. J.; Daugherty, W. H.; Gottleib, Leo; Scott, W. S., and McBryde, C. M.: Radioactive iodine therapy of hyperthyroidism: determination of optimal dosage, Southern Medical Journal 44:648 (July) 1951.

32. Werner, S. C.; Quimby, E. H., and Schmidt, C.: The clinical use of radioactive iodine, Bull. New York Acad. Med. 24:549 (Sept.) 1948.

33. Werner, S. C.; Quimby, E. H., and Schmidt, C.: The use of tracer doses of radioactive iodine, I^{131}, in the study of normal and disordered thyroid function in man, J. Clin. Endocr. 9:342 (April) 1949.

34. Werner, S. C.; Quimby, E. H., and Scbmidt: Radioactive iodine, I^{131}, in the treatment of hyperthyroidism, Am. J. Med. 7:732 (Dec.) 1949.

35. Williams, R. H.; Tower, B. T.; Jaffe, H.; Rogers, W. F., and Tagnon, Rene: Radiotberapeusis, Am. J. Med. 7:702 (Dec.) 1949.

36. Fermi, Enrico: Radioactivity induced by neutron bombardment, Nature, 133:757 (May) 1934.

Patronize JOURNAL advertisers.

UNIVERSITY OF KANSAS SCHOOL OF MEDICINE

Postgraduate Medical Courses

PEDIATRICS

March 15, 16 & 17, 1954

Guest Instructors:

RUSSELL J. BLATTNER, M.D., Professor of Pediatrics and Chairman of Department, Baylor University College of Medicine, Houston.

J. ROSWELL GALLAGHER, M.D., Chief, Adolescent Unit, The Children's Medical Center, Harvard University Medical School, Boston.

C. EVERETT KOOP, M.D., Assistant Professor of Pediatric Surgery, University of Pennsylvania School of Medicine; also Pediatric Surgeon, The Children's Hospital, Philadelphia.

Subjects to be Discussed:

Changing Concepts of Pediatric Surgery.
Certain Aspects of Squint.
Urinary Tract Infections Resistant to Antibiotic Therapy.
Responsibility of Physician in Emergency Treatment of Burns.
Common Foot Problems in Children.
Present Attitude Toward Tonsil and Adenoid Problem.
Surgical Emergencies in the Neonatal Period.
General Problems of Growth and Development.
Sexual Development of the Female Child.
Dental Problems of the Adolescent.
Adolescent Acne Vulgaris.
The Adolescent Conscience.
The Juvenile Delinquent.
Retrolental Fibroplasia; New Development in Etiology and Prevention.
Etiologic Factors in Enteric Infections.
Prevention of Recurrent Rheumatic Fever.
The Use of Gamma Globulin.
Treatment of Enuresis.
Increasing Importance of Tuberculin Skin-Testing in Children.
Recent Developments in Prevention of Poliomyelitis.

Case Presentations: Atypical Appendicitis; Patent Ductus in Infancy; Premature Closure of Sutures; Volvulus; The Obese Pre-adolescent Child; Retarded Growth; Lead, Iron & Mercury Poisoning; Encephalitis.

This will be followed by a three-day course on PUBLIC HEALTH AND PREVENTIVE MEDICINE, **March 17, 18 & 19**, with the Wednesday program arranged jointly for the two groups attending these courses.

PERIPHERAL BLOOD AND BONE MARROW STUDY

March 11 & 12, 1954

A two-day session in actual microscopic examination of peripheral blood and bone marrow preparations of various hematologic diseases. Also, colored photographic slides will be projected for study. Ample time will be allotted for question and answer sessions.

Faculty:

Sloan J. Wilson, M.D., Associate Professor of Medicine.
Kurt R. Reissmann, M.D., Associate Professor of Medicine. And Assistants.

RADIOLOGY

February 15, 16 & 17, 1954

NURSING

February 8, 9 & 10, 1954

always consider ERYTHROCIN

A SELECTIVE ANTIBIOTIC



ORALLY EFFECTIVE

against staphylococci, streptococci and pneumococci—especially indicated when patients are allergic to other antibiotics or when the organism is resistant.



A DRUG OF CHOICE

against staphylococci—because of the high incidence of staphylococci resistant to other antibiotics.



A DRUG OF CHOICE

because it is less likely to alter normal intestinal flora than other antibiotics, except penicillin; gastrointestinal disturbances rare; no serious side effects reported.



USE ERYTHROCIN

in pharyngitis, tonsillitis, otitis media, sinusitis, bronchitis, scarlet fever, pneumonia, erysipelas, pyoderma and certain cases of osteomyelitis.



DOSAGE

average adult dose is two 100-mg. tablets every four to six hours. Specially-coated ERYTHROCIN tablets are available in bottles of 25 and 100. Abbott

3-234

Trade Mark ERYTHROMYCIN, ABBOTT CRYSTALLINE

THE KANSAS PRESS LOOKS AT MEDICINE

Editor's Note. In this section the JOURNAL *reproduces editorials relating to medicine which have appeared in the lay press. An effort is made to include both favorable and unfavorable comments, and the Editorial Board in no instance assumes responsibility for the opinions expressed.*

APPENDIX FOR SALE

Medicine is a profession, not a merchandising service. Thus doctors have traditionally made varying charges for essentially similar operations and treatments according to such factors as individual prestige, wealth or poverty of the patients, and specific difficulties or complications that may be encountered.

For that reason, many of them are looking with more and more of a jaundiced eye at the medical and surgical insurance plans which set maximum payoffs for specified services.

It is true that these are only agreements between insurance companies and insured. There is nothing to keep doctors from charging their own price and collecting the difference from the patient. And they do it in thousands of instances.

But the pressure to conform to insurance schedules is becoming almost irresistible in places like Akron, Ohio, where group insurance plans cover the entire rubber industry and therefore almost the entire working population.

If the schedule says $100 for an appendix operation, and if the majority of doctors are charging $100 to middle-class patients, the upper crust patients can see no reason why they should pay more.

Nor can the man who has "complications." The doctor has plenty of easy, normal cases, hasn't he? Why can't he take a few tough ones in stride without tacking on an extra charge?

He doesn't have the credit losses he used to, does he? Then why should he charge the rich more to make up for a mythical "poor" which he no longer has?

These are hard questions to answer. Yet there is validity in the complaint that in areas where group insurance is almost universal, doctors tend to be forced into a straightjacket that bears a startling resemblance to socialized medicine.

In fact, it can almost be charged that it is social-

ACCIDENT, HOSPITAL AND SICKNESS INSURANCE

For Physicians, Surgeons and Dentists Exclusively



$5,000 accidental death Quarterly $8.00 $25 weekly indemnity, accident and sickness	$15,000 accidental death Quarterly $24.00 $75 weekly indemnity, accident and sickness
$10,000 accidental death Quarterly $16.00 $50 weekly indemnity, accident and sickness	$20,000 accidental death Quarterly $32.00 $100 weekly indemnity, accident and sickness

Cost Has Never Exceeded Amounts Shown

ALSO HOSPITAL INSURANCE

	Single	*Double*	*Triple*	*Quadruple*
60 days in Hospital	5.00 per day	10.00 per day	15.00 per day	20.00 per day
30 days of Nurse at Home	5.00 per day	10.00 per day	15.00 per day	20.00 per day
Laboratory Fees in Hospital	5.00	10.00	15.00	20.00
Operating Room in Hospital	10.00	20.00	30.00	40.00
Anesthetic in Hospital	10.00	20.00	30.00	40.00
X-Ray in Hospital	10.00	20.00	30.00	40.00
Medicines in Hospital	10.00	20.00	30.00	40.00
Ambulance to or from Hospital	10.00	20.00	30.00	40.00
COSTS (Quarterly)				
Adult	2.50	5.00	7.50	10.00
Child to age 19	1.50	3.00	4.50	6.00
Child over age 19	2.50	5.00	7.50	10.00

$4,000,000.00 INVESTED ASSETS

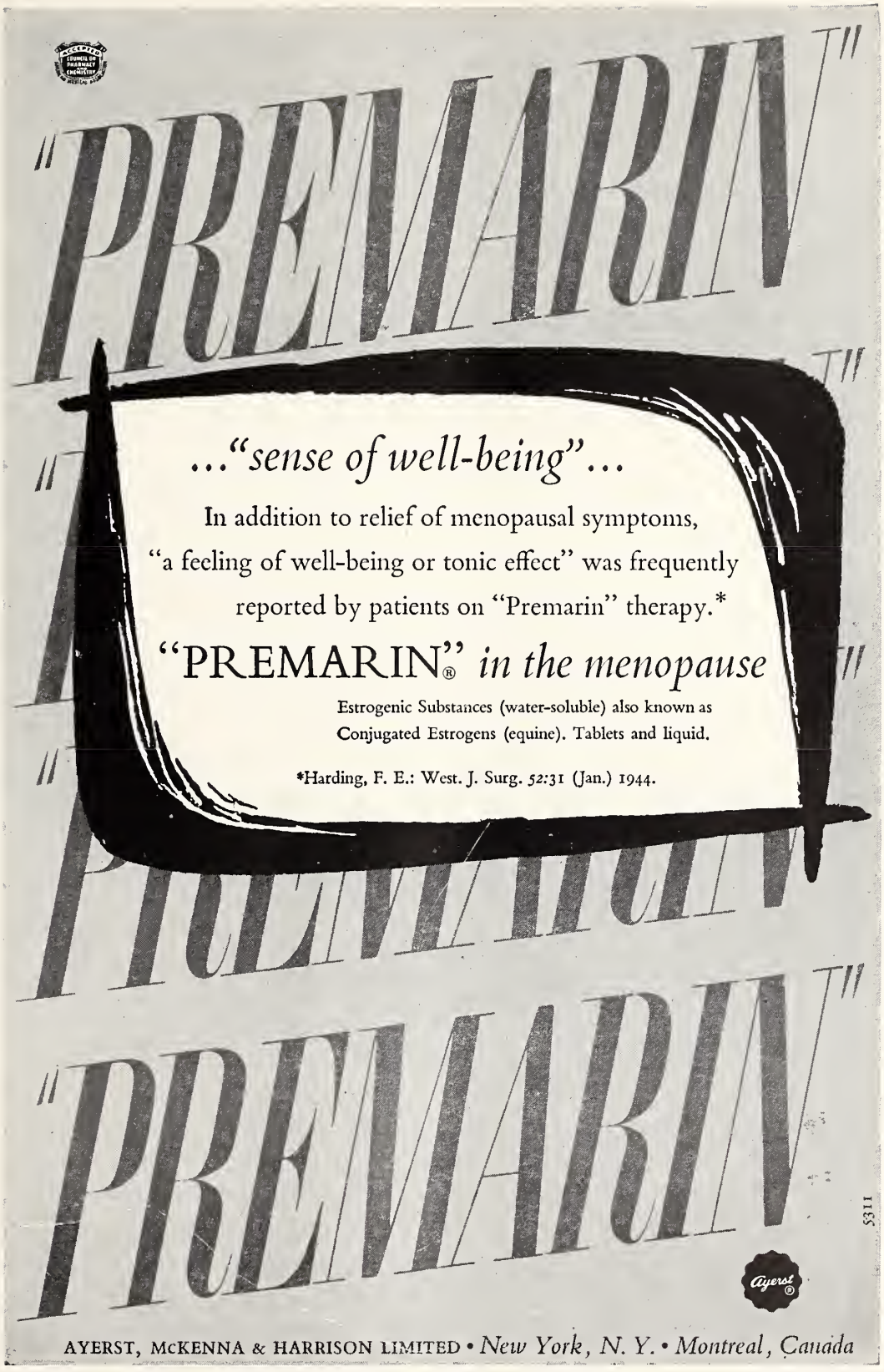
PHYSICIANS CASUALTY ASSOCIATION
PHYSICIANS HEALTH ASSOCIATION

$19,500,000.00 PAID FOR CLAIMS

51 years under the same management

400 First National Bank Building Omaha 2, Nebraska

$200,000.00 deposited with State of Nebraska for protection of our members


"PREMARIN"
...*"sense of well-being"*...
In addition to relief of menopausal symptoms,
"a feeling of well-being or tonic effect" was frequently
reported by patients on "Premarin" therapy.*
"PREMARIN"® *in the menopause*
Estrogenic Substances (water-soluble) also known as
Conjugated Estrogens (equine). Tablets and liquid.
*Harding, F. E.: West. J. Surg. 52:31 (Jan.) 1944.
5311
Ayerst
AYERST, McKENNA & HARRISON LIMITED • *New York, N. Y.* • *Montreal, Canada*

ized medicine. Because when its cost is paid as a deductible expense by a corporation, the government stands half of it in tax loss.

But it's here. It's getting bigger every day. And by and large, the benefits are tremendous compared to the drawbacks.

The horse and buggy days of doctoring are gone. And some of the horse and buggy methods of charging for a doctor's service are just going to have to go along with them.—*Iola Register, November 16, 1953.*

Gift From Tuberculosis Association

A gift of $10,000 a year for five years to the new chair of thoracic diseases at the University of Kansas School of Medicine was announced recently by the Kansas State Tuberculosis Association. Each annual contribution will represent proceeds from the sale of 1,000,000 Christmas seals.

Dr. Martin James FitzPatrick, who joined the faculty of the medical school two months ago as assistant professor of medicine, will direct the work of the new chair. He is a diplomate of the American Board of Internal Medicine, an associate fellow of the American College of Chest Physicians, and a member of the American Trudeau Society and of the American Federation for Clinical Research.

The New England Medical Journal recently reported that one doctor who carefully surveyed his mail over a 12-months period, May 1, 1952, to April 30, 1953, received a total of 3,305 pieces of direct mail advertising. Of this total, 2,883 pieces were from pharmaceutical manufacturers. Products used in the treatment of cardiovascular disease headed the list, with antibiotics and sulfonamides next.

Demand for A.M.A. radio transcriptions on common health subjects hit an all-time high during 1953, according to a recent release from association headquarters. More than 700 sets were distributed for broadcasting over local radio stations in all parts of the country. Assisting the Bureau of Health Education in distributing these health education platters and promotional material are 14 distribution centers set up by state medical societies.

The transcriptions are provided without charge by the A.M.A. as a public service. Requests from groups not associated with the medical profession, such as universities and high schools, are always cleared through the local medical society.

The 1954 meeting of the American Medical Association will be held at San Francisco, June 21-25.

ANNUAL CLINICAL CONFERENCE

CHICAGO MEDICAL SOCIETY

March 2, 3, 4, 5, 1954

Palmer House, Chicago

DAILY HALF-HOUR LECTURES BY OUTSTANDING TEACHERS AND SPEAKERS on subjects of interest to both general practitioner and specialist.

PANELS ON TIMELY TOPICS.

MEDICAL COLOR TELECASTS.

TEACHING DEMONSTRATIONS.

SCIENTIFIC EXHIBITS worthy of real study and helpful and time-saving TECHNICAL EXHIBITS.

The CHICAGO MEDICAL SOCIETY ANNUAL CLINICAL CONFERENCE should be a MUST on the calendar of every physician. Plan now to attend and make your reservation at the Palmer House.



"...when the patient is in acute distress from waterlogging..."

"Meralluride sodium solution (MERCUHYDRIN) in 1 to 2 cc. doses intramuscularly has been very effective and is not painful."* In acute congestive failure, MERCUHYDRIN characteristically curbs tissue inundation and relieves dyspnea, orthopnea and cardiac asthma.

Ampuls of 1 cc., 2 cc., and 10 cc. vials.

*Stead, E. A., Jr., in Cecil, R. L., and Loeb, R. F.: Textbook of Medicine, ed. 8, Philadelphia, W. B. Saunders Co., 1951, p. 1065.

MERCUHYDRIN®
(meralluride injection U.S.P.)

Leadership in diuretic research
Lakeside LABORATORIES, INC., MILWAUKEE 1, WISCONSIN

BOOK REVIEWS

Respiratory Diseases and Allergy. By Josef S. Smul. Published by Medical Library Company, New York. 80 pages. Price $2.75.

In this book the author attempts to cover some of the major diseases of the organs comprising the respiratory system in the incredible space of 52 pages of text. In his preface he claims to be able to do this in a complete manner, but never quite achieves this end. As is usually the case in this type of endeavor, a "new approach" is heralded as the magic formula which attempts to reduce the multiple problems to fit the limited confines of this book.

Dr. Smul is able to break the complex interplay of infection and allergy into six compartments. He mentions the word allergy repeatedly but fails to define it. He coins new words which fail to contribute to a better understanding of fundamental problems. There is only one reference noted in the entire text. There is no mention of the extensive experimental work that has been done in this broad field. Clinical material has not been used in a recognizable form.

The author should have indicated the level of medical knowledge he was aiming at, in writing this book. The factual inaccuracies and uncompromising tone of presentation of the subject matter limit the scope and usefulness of this book to the student or to the practitioner.—*M.J. FP.*

Diseases of Women. By R. J. Crossen. Published by C. V. Mosby Company, St. Louis. 935 pages, 940 illustrations, 41 colored illustrations. Price $18.50.

This book is a revised edition of a long established text and reference book in which medical gynecological problems are clearly and systematically covered.

The chapter arrangement and method of material presentation are essentially unchanged. Many of the chapters have only minor revisions; more extensive changes consist mainly of adding or revising sections within chapters.

Some of the phases of gynecology new in this text are sections on the controversial subjects of intraepethelial carcinoma and factors predisposing to carcinoma; the psychosomatic aspect of puberty and adolescence as a method of preventing later psychosomatic problems; and embryology of the ovary as it is related to oogenesis and development of various ovarian structures.

The section on radiation therapy of cervical carcinoma is completely rewritten by Dr. A. N. Arneson.

QUESTION?

Have you enrolled in the KANSAS MEDICAL SOCIETY Approved DISABILITY INCOME PLAN?

- **Endorsed and Supervised by Your Society**
- **Non-Cancellable Features**
- **Lifetime Accident Benefits**
- **Five-year Sickness Benefits**
- **30% to 50% Saving in Cost**
- **Over 65% of Eligible Membership Now Enrolled**

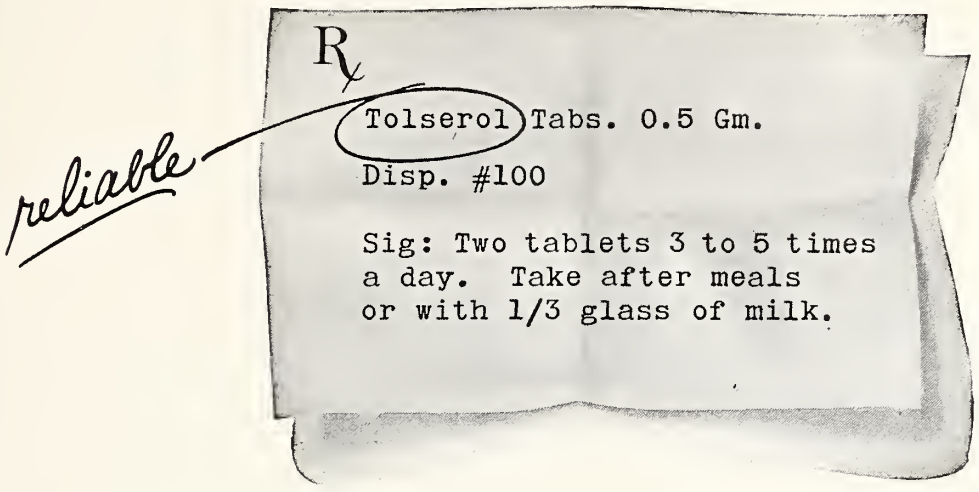
Officially approved and endorsed by the Council and the House of Delegates.

For Complete Information Write—Kansas Medical Society, 315 West Fourth, Topeka, Kansas

or

FIRST INSURANCE AGENCY
Washington National Insurance Company
639 New England Building Topeka, Kansas

RAPID ABSORPTION—*MAXIMUM THERAPEUTIC EFFECT*



The clinical effectiveness of different brands of mephenesin tablets depends on their rate of absorption. A mephenesin tablet that disintegrates slowly is absorbed slowly. The resulting low blood levels may never produce a maximum therapeutic effect. Results with such a tablet are usually poor.

Tolserol Tablets are a result of extensive study and are formulated to disintegrate rapidly for fast absorption, thus maintaining optimum blood levels.

Tolserol

(*Squibb Mephenesin*)

Complete information on the use of Tolserol in muscle spasm of rheumatic disorders, in neurologic disorders and in acute alcoholism is available from the Professional Service Department, Squibb, 745 Fifth Avenue, New York 22, N. Y.

SQUIBB

In this section basic physics and its practical application in radiation therapy is clearly and concisely explained.

Dr. Willard Allen not only wrote an excellent section on "Endocrine Relations Concerned in the Ovarian Cycle," but also in other sections made valuable additions on the therapy of endocrine disturbances.

The usual excellent chapter on "Gynecological Examination and Diagnosis" is included. In this chapter the steps and techniques of taking a proper gynecological history and making an adequate examination are outlined in detail. Also in this chapter special examinations and new diagnostic aids such as culdoscopy, culdocentesis, vaginal smears, tests for ovulation, and determining the exact day of ovulation and new pregnancy tests are added.

The chapter on "The Diseases of the Ovary and Parovarium" is revised with particular emphasis placed on grouping those related in origin and presenting the newer concepts concerning the origin of ovarian tumors.

This edition of an excellent text maintains the same high standards that have been associated with it since the first edition appeared in 1907 by Dr. H. S. Crossen.—*W.J.S.*

Patronize JOURNAL advertisers.

BOOKS NEEDED IN ISRAEL

An appeal for medical books published since 1940 was issued recently in an effort to relieve a shortage of such volumes in hospitals, universities, and offices of physicians in Israel. The United States State Department, sponsor of the project, will provide funds for shipment of the volumes from New York to Israel. Donors are asked to send contributions to Books for Israel, 115 King Street, New York 1, New York.

The past 50 years have been momentous in the fight against tuberculosis. At the outset it was found that the control of the disease was the problem both of the physician and the layman. . . . The achievements have been substantial. Our horizon has been raised to new and challenging vistas beyond. Despite our gains, we still are dealing with a serious killing disease that has sacrificed countless lives that should have been preserved for productive effort. So far as we can see at this time, tuberculosis will be with us for many decades to come. But we are drawing nearer to a complete understanding of its cause and control, which is beyond the tubercle bacillus alone. When we learn how to maintain our factors of resistance at a level that will keep the infection dormant, we may achieve as practical a result as



Woodcroft Hospital--Pueblo, Colorado

A private hospital for the scientific treatment of neuro-psychiatric disorders, including alcoholism and drug addiction. Beautiful landscaping and home-like surroundings afford a restful atmosphere. Accommodations vary from single rooms with or without bath to rooms en suite, allowing for segregation of guests.

Detailed information furnished on request.

KARL J. WAGGENER, M.D.

atopic dermatitis...

Cortef*

acetate ointment

In 5 Gm. tubes of 1.0% and 2.5% concentration

*Trademark for Upjohn's brand of hydrocortisone (compound F)

Upjohn *The Upjohn Company, Kalamazoo, Michigan*

complete eradication of the tubercle bacillus and the clinical manifestations of the germs in our bodies.—*H. R. Edwards, M.D., American Review of Tuberculosis, March, 1952.*

"We speak of medicine as both a science and an art, and surely these two aspects are complementary. Science is analytic, explaining, or seeking to explain, the whole in terms of the part; art is intuitive, and sees in the whole something more than can be explained as the sum of its parts. Alike in its comprehensiveness, its balance and its insight, the medicine which we have inherited from the great masters of the past in unique; and if we remain true to its philosophy we need have no fears for its future."—*Sir Russell Brain in the Lancet, quoted in Westchester Medical Bulletin, July, 1953.*

K.M.S. annual meeting, May 2-6, 1954, Topeka.

CLASSIFIED ADVERTISEMENTS

FOR SALE—Stock of medicines by physician wishing to retire after 39 years in same location. Will sell or rent office building and equipment, including x-ray. Small town in central Kansas. Two fine hospitals within 13 miles. Eight towns without physicians within 14 miles. Write the Journal 14-53.

FOR SALE OR LEASE—Seven-room office, newly decorated and equipped, in busy shopping center near large housing area and industries, large city, central Kansas. Write the Journal 15-53.

WANTED TO BUY—a good used optical trial case. In reply quote price. Write the Journal 16-53.

FOR SALE—252½ mg. of radium in containers of varying sizes. Write the Journal 1-54.

WANTED—Registered medical technician, male or female, to work in a clinic of 12 doctors and in a midwestern city of 100,000 population. Excellent working conditions, no night calls. Write the Journal 2-54.

FOR SALE—Spencer monocular microscope, like new. Two eyepieces 10x and 6x; three objective lenses 10x, 44x and 95x oil immersion; mechanical stage: substage light condenser and individual lamp; carrying case. Excellent for medical student or physician's laboratory. Price $225. Write the Journal 3-54.

Nationally advertised Surgical Supplies and Equipment have been placed at Topeka, Joplin, Kansas City and St. Joseph for your convenience by—

GOETZE NIEMER COMPANY

Management by Dr. W. F. Goetze, member of the American Medical Ass'n, assures intelligent servicing of your orders.

It's Convenient to Buy Kanox

THERAPEUTIC OXYGEN AND ANESTHETIC GASES

Oxygen Therapy
Regulators and Equipment
Repair Service for
Regulators, Flowmeters
and Humidifiers



Nitrous Oxide
Ethylene
Carbon Dioxide
Helium
Oxygen
Cyclopropane

KANSAS OXYGEN, Inc.

Phone 5-5551 HUTCHINSON, KANSAS Box 551

Since its introduction over four years ago, Chloromycetin has been used by physicians in practically every country of the world. More than 11,000,000 patients have been treated with this important antibiotic—

world's outstanding therapeutic agents.



TABLE OF CONTENTS

FEBRUARY, 1954

ORIGINAL ARTICLES

Modern Concept of Otitis Media—Harold L. Hickey, M.D., Denver, Colorado . . . 63

The Pathology of Ectopic Pregnancy—Fred A. Brandt, Kansas City, Kansas 66

Weber-Christian Disease: A Case Report—Charles A. Pokorny, M.D., and C. Alexander Hellwig, M.D., Halstead, Kansas . . . 70

Addison's Disease Complicated by Pregnancy: A Case with Postpartum Hypertension—Thomas J. Luellen, M.D., and Ray A. West, M.D., Wichita, Kansas 73

EDITORIALS

Prehistoric Medicine 79

Events in Washington 80

Doctors at War and at Home 80

Memorial Fund 81

MISCELLANEOUS

President's Page 78

Clinicopathological Conference 85

The Cerebral Circulation: Comments on Cerebral Angiospasm—Senior Thesis . . . 92

Entered as second-class matter at the post office at Fulton, Missouri, June 19, 1953, under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of October 3, 1917, authorized on July 2, 1918. Office of Publication: 1201-05 Bluff Street, Fulton, Missouri.

THE JOURNAL OF THE KANSAS MEDICAL SOCIETY

Editorial Board

ORVILLE R. CLARK, M.D., *Editor,* Topeka
JOHN W. CAVANAUGH, M.D., Topeka
DAVID E. GRAY, M.D., Topeka
RICHARD GREER, M.D., Topeka
DWIGHT LAWSON, M.D., Topeka

Associate Editors

DONALD P. TREES, M.D., Wichita
GLEN R. SHEPHERD, M.D., Kansas City

Information

Ownership: The Journal is a non-profit publication owned and published monthly by the Kansas Medical Society.

Subscription: A year's subscription to the Journal is included in membership in the Kansas Medical Society, with $2.00 of each member's dues apportioned to the Journal. Rates to others, except in foreign countries, $4.00 per year or 60c per copy.

Material: Scientific articles, editorials, and data of general interest are invited from all members. Articles are to be submitted on condition that they are contributed solely to this publication. A right is reserved to reject any material deemed unsatisfactory.

Manuscripts: Only manuscripts that are typewritten on one side, double spaced, and original copies can be accepted. Manuscripts will be returned upon request.

Advertising: All advertising contracts, and all copy from advertisers under contract are subject to approval of the editorial board. Copy should be received by the 20th of the month immediately preceding the month of publication.

Non-Responsibility: Although an effort is made to publish only accurate articles and legitimate advertisements, the Journal denies legal responsibility for any statements, opinions, or advertisements appearing under the names of contributors or concerns.

PAULINE FARRELL, Managing Editor and Advertising Manager

OLIVER E. EBEL, Business Manager

THE KANSAS MEDICAL SOCIETY

President.....LUCIEN R. PYLE, M.D., Topeka
Past President....WARREN F. BERNSTORF, M.D., Winfield
President-Elect.....MURRAY C. EDDY, M.D., Hays
First Vice-President..JOHN M. PORTER, M.D., Concordia
Second Vice-President..CLYDE W. MILLER, M.D., Wichita
Constitutional Secretary..DALE D. VERMILLION, M.D., Goodland
Treasurer.....JOHN L. LATTIMORE, M.D., Topeka
AMA Delegate 1953-1954, LAURENCE S. NELSON, M.D., Salina
AMA Delegate 1954-1955, GEORGE F. GSELL, M.D., Wichita

Councilors and Councilor Districts

First District.....W. L. ANDERSON, M.D., Atchison
Second District .....H. P. JONES, M.D., Lawrence
Third District....CHARLES E. VESTLE, M.D., Humboldt
Fourth District.....F. C. TAGGART, M.D., Topeka
Fifth District.....HAROLD M. GLOVER, M.D., Newton
Sixth District.....NORTON L. FRANCIS, M.D., Wichita
Seventh District..SEVERT A. ANDERSON, M.D., Clay Center
Eighth District.....A. E. RUEB, M.D., Salina
Ninth District.....JAMES L. JENSON, M.D., Colby
Tenth District.....L. W. REYNOLDS, M.D., Hays
Eleventh District.....C. V. BLACK, M.D., Pratt
Twelfth District.....R. G. KLEIN, M.D., Dodge City

OLIVER E. EBEL, Executive Secretary

RUEBEN M. DALBEC, Executive Assistant

Executive Office: 315 West Fourth Street, Topeka

THE JOURNAL
of the
KANSAS MEDICAL SOCIETY
Copyright, 1954, by The Kansas Medical Society

Volume LV FEBRUARY, 1954 No. 2

Modern Concept of Otitis Media

Harold L. Hickey, M.D.
Denver, Colorado

The title of this presentation might lead some of this audience to believe that the writer is about to regale you with accounts of newly discovered diseases. This, of course, would be impossible as descriptions of otitis media date back to writings of Hippocrates. However, in recent years certain potent factors have modified our thinking and would seem to warrant a studied assessment of ideas and conclusions in the light of new contributions to an old subject. Although acute and chronic otitis media might be considered merely phases of the same condition, it seems profitable to consider them separately.

The pathogenesis of acute otitis media is generally accepted to be the extension of upper respiratory infections, exanthemata, tonsillitis, influenza, so-called virus infections, etc., via the eustachian tube to the middle ear. Anatomical differences in the tube between children and adults favor a greater incidence in the former. Streptococcus hemolyticus still holds first place in bacteriology, but staphylococcic, pneumococcic, and influenzal infections are not uncommon, while Friedlander bacillus and Klebs-Loeffler are rather rare.

The symptoms of otalgia, conduction deafness, fever, leucocytosis, and varying prostration are well known. Local redness of the tympanic membrane, obliteration of land marks, bulging, or even spontaneous rupture with purulent drainage may occur within a few hours of onset. Clinicians (especially otologists) should know that periauricular and mastoid tenderness often seen early is merely part of the invasive stage and not due to mastoiditis. It usually disappears with adequate drainage.

All of the above sounds familar, but now the picture changes. Incident to the universal use of antibiotics or chemotherapy, the fulminating febrile stage appears controlled; and, unless we are alert, subsequent events may go unnoticed. The majority of pediatricians and general practitioners, yes even otologists, are lulled into a sense of satisfaction that the disease is cured merely because sufficient time and effort are not put forth to study in detail the ears and nose. A careful history and follow-up of these patients is also essential.

Meticulous cleansing of the auditory canal and inspection of the membrana tympani, even when drainage is not present or has ceased during the dramatic antibiotic blitz, may reveal a dull, lusterless but not inflamed membrane, hearing subnormal, and mobility impaired when checked with the Siegel pneumatic scope. Infections in the nose or sinuses, enlarged adenoid masses even after tonsil and adenoid operations, may contribute to the failure of resolution. However, probably the most common error is the failure to establish drainage by myringotomy in the acute stage as we would have done years ago. It is a cardinal principle of good surgery that infections require drainage, and the galaxy of modern drugs, wonderful as they are, has not changed that principle.

Patients, especially children, frequently present recurrent attacks which are interpreted as new infections when more than likely the original infection was not completely controlled due to discontinuation of medication too soon or failure to perform myringotomy when it was needed.

It is becoming standard procedure in most of the city and town schools, as well as in many rural areas, to make periodic hearing tests by audiometer. While

Presented before the Eye, Ear, Nose and Throat Section of the Kansas Medical Society, May 6, 1953.

this is only a screening test which should lead the patient to the otologist, its value cannot be questioned. It is my opinion that many of the hearing defects thus discovered might have been prevented by adequate care of acute otitis media.

From the above remarks I would not care to leave the impression that every case of acute otitis media requires myringotomy, but it is a statistical fact that mastoiditis occurs in a higher percentage of patients in whom the drum ruptures spontaneously. Also, not all patients require antibiotics or chemotherapy. Nasal shrinkage to encourage ventilation of the middle ear, having the patient rest with the involved ear uppermost, management of allergies, and instruction as to nose blowing all have a bearing on the outcome of the acute ear.

Contrary to popular belief, simple mastoidectomy is still necessary in the management of some of the neglected and ill treated cases; however, incidence is so much lower that it is becoming difficult for residents in otolaryngology to have adequate training in performing the operation. I still perfer the postauricular approach for simple mastoidectomy as it offers a better opportunity, for me at least, to manage extensions of the suppurative process. Many of us have seen ears with mastoids successfully operated upon but with partially stenosed auditory canals and severely depressed scars. With a little care in technique at operation and adequate postoperative packing of the canal, such results can be avoided.

It should be pointed out that, due to antibiotic suppression, mastoid infection may develop and progress almost to complete breakdown without alarming symptoms. It may develop without pain, fever, or increased white count. Failure of the tympanic membrane to return to normal and the lack of complete restoration of good hearing should excite suspicion. Unfortunately, a child in school does not always report decreased hearing, but an alert teacher or parent may detect it.

Lack of adequate treatment encourages chronicity, and this is especially true in the case of a spontaneous rupture of the tympanic membrane in Shrapnell's area. The chronic ear will be more fully discussed later.

There is a condition which occupies a middle ground between acute and chronic otitis media and about which much has been said and written. I refer to otitis media with effusion, secretory otitis media, hydrops of the middle ear, aerotitis media, nonsuppurative or catarrhal otitis media. The particular terminology is not important as long as we understand the mechanism of its occurrence and recognize the symptoms. In my personal experience, it has been a disease of adults more frequently than of children. An airplane trip during an upper respiratory infection, especially in a non-pressurized plane, is a common etiologic incident. In the presence of a blocked eustachian tube, equalization of external and intratympanic pressure is impossible, a negative pressure in the middle ear causes pain and, in many cases, the pouring out of fluid of a serous type. The result is rather sudden conductive deafness. The patient may describe a sensation of shifting fluid in the ear.

When the condition is sudden and severe enough to cause redness or even hemorrhagic changes in the tympanic membrane, attention is drawn to the condition and proper treatment will probably ensue. However, in those cases of milder barotrauma or symptoms of fullness and slight deafness incident to upper respiratory infection without the airplane factor, diagnosis may not be so apparent. I have listened to many speakers and have read many articles describing the "amber color of the drum," the "chalky appearance of the umbo," the shifting fluid line, and bubbles on inflation, and yet when I am faced with a diagnosis in a suspected case I often fail to find any of the described findings. In the presence of conductive deafness of recent origin and suggestive symptoms as noted by the patient, I have found the most reliable finding to be immobility of the tympanic membrane when viewed through the Siegel scope. One pathognomonic sign is the clearing of the impaired hearing when the head is tilted forward or to the opposite side, only to have the impairment recur when a normal head position is assumed. It is probable that the fluid overlying the round window causes the impairment.

Treatment in years past has been on the conservative side, such as nasal shrinkage, eustachian catherization and inflation, etc., none of which produced results. Only in recent years has it been appreciated that evacuation of the fluid from the middle ear is the answer. This is accomplished either by paracentesis in the posterior inferior quadrant and aspiration of the viscid stringy fluid with a small suction canula, or by needle puncture and withdrawal of the fluid with a small attached syringe. Since the drum is not inflamed, many workers use no anesthesia. However, the average patient is fearful of having his ear "lanced," and I find that the use of a small amount of local topical anesthesia is reassurring and makes for better co-operation.

In cases of short duration, a single aspiration usually suffices, and the patient is delighted with the almost instantaneous relief of pressure and restoration of hearing. In long standing cases which may not have been previously recognized, repeated aspirations may be needed. This is especially true in persons with chronic allergies who have habitually sniffed through the nose forcibly many times a day. The small tympanic opening heals quickly and causes little discomfort.

In considering chronic suppurative otitis media we have a disease which is age old. There are many possible ramifications and complications to consider, but primarily in diagnosis these narrow down to a few important findings. Location of the perforation tells a great deal, and on that basis we may divide our cases into non-dangerous and dangerous types. The central perforation with mucopurulent discharge, usually odorless, is found in patients with intermittent symptoms, invariably subject to exacerbation during a cold. Hearing will be impaired but may be found to be remarkably good if the patient is tested during the dry phase. This is the non-dangerous type.

By contrast, the marginal perforation, especially in Shrapnell's area, or the so-called attic perforation, from which discharge is more or less continuous and malodorous, should be classified as the dangerous type. The drainage may be almost caseous at times, and extraction with ether may show under the microscope cholesterin crystals characteristic of a cholesteatoma. Inasmuch as the pathology centers around the epitympanum as well as the middle ear proper, the ossicular chain will be damaged, and hearing is consistently worse than in the non-dangerous type.

Since either condition is of long standing, x-ray films of the mastoid will show sclerosis and obliteration of most of the pneumatic structure; the anterior wall of the lateral sinus will stand out in sharp contrast. I get more information from a well taken Law position view, but others prefer a Stenver's position. Dr. Gilbert Owen in Los Angeles has devised a special projection which, in his hands, gives a remarkable delineation of the attic, antrum, and even semicircular canals. However, it does not seem to be usable by most other radiologists. In the case of cholesteatoma which is sizable, there will be seen a rarefied area within the sclerotic antrum or along the lateral sinus wall, which is an important aid to diagnosis.

The presence of aural polypi or bleeding granulations protruding through the tympanic perforation also suggests dangerous pathology. Symptoms of temporal headache on the involved side, dizziness, nystagmus, nausea, and vomiting emphasize the seriousness of the condition. Occasionally a sudden facial paralysis may indicate extension of suppuration, and I have seen a few patients who had postponed advised surgery become emergency problems with otitic meningitis or other intracranial complications.

Accurate evaluation of symptoms and signs leads to a diagnosis and determines whether treatment can be conservative and watchful (non-dangerous type) or whether surgery is imperative (dangerous type).

In the conservative treatment, toilet of the ear to keep it clean and dry, and avoidance of swimming and other exposures to water in the ear is important. The patient should realize that prompt care of respiratory infections may avoid recurrent ear attacks. Conservation of hearing is always important, but since some irreparable damage has usually taken place, all we can hope for is to prevent further loss.

For several years at least one author (and perhaps others) has emphasized the importance of chronic antral infection in relation to chronic otitis media, and I am sure we have all noted ipsolateral involvement. Granted that something is gained by proper care, including surgery on the sinus condition, I can hardly accept the glowing reports of cessation of aural drainage, healing of large perforations, and restoration of normal hearing following radical antrum operations.

Local drug therapy has been discussed in a previous paper. In addition to boric alcohol drops when tolerated, or Furacin anhydrous solution, I find many patients benefitted by insufflation of Sulzberger Iodo-bor powder. In cases of central perforations, after they are dry, closure can often be effected by careful use of trichloracetic acid to the edge of the perforation. No attempt should be made to close an upper marginal perforation. If the patient will follow the advice, keeping the involved ear turned upward during sleep will discourage the capillary seepage from the nasopharynx which causes recurrent drainage.

Aural polyps should be removed, using care to avoid snare traction on the pedicle which may be attached to the middle ear promontory. Granulations should be cauterized or removed with a sharp curet or middle ear cupped forceps. Attic irrigations using a small special canula and a dehydrating solution may be tried in those borderline cases which have not shown definite evidence of surgical indications.

One of the problems which I find especially difficult is the decision as to proper treatment, especially surgical, when dealing with an individual with bilateral chronic otitis media. Given a patient with seriously impaired hearing in one ear and continuing evidence of chronic suppuration in the opposite ear, the added social and occupational hazard which would result from a radical operation on the better hearing ear poses a difficult decision. In general, we should be guided by clear indications just as though the patient had a good hearing ear on which to depend.

A few years ago I was forced to perform endaural radical mastoidectomy on both ears in a young man of 17 because of extensive cholesteatoma with impending intracranial involvement. Fortunately, dry ears were secured but with a reduction in hearing bilaterally to loud spoken voice at one foot from the ears. However, he was fitted with a bone conduction

unit (rare in my experience), and he was able to hear ordinary conversation at 20 feet without seeing the speaker's lips. In another case, a school teacher, I debated some time as to a needed radical operation because the opposite ear was not a usable hearing ear. She was operated upon and carries on very successfully, also by the aid of a bone conduction unit.

Having made a diagnosis of chronic suppurative otitis media of the dangerous type which appears to demand operation, both in an attempt to stop the suppuration and to obviate the probability of intracranial extension, in other words to make it a safe ear, how should we approach it? The writer has been in practice long enough to pass through the period when both simple and radical operations were relatively common. His early training and experience were in the post-auricular approach. However, when one has become accustomed to the endaural approach and realizes the many advantages to the operator, the patient, and the ever present need to reduce hospitalization time, I feel sure that he would never wish to return to the post-auricular method for modified radical or complete radical mastoidectomy. The work of Lempert, Meltzer, House, and many others is familiar to most of you, and, if you are not now using the endaural approach, I highly recommend that you take steps in preparation to do so.

Detailed description of the surgical technique of endaural mastoidectomy would serve no useful purpose at this time, but since the operative field is a highly specialized one intimately related to such vulnerable structures as the facial nerve, horizontal semicircular canal, middle fossa dura, oval and round windows, it should be apparent that only a well trained otologist should attempt the operation. Immediate skin grafting of the middle ear and radical cavity encourages more rapid healing, but this is not absolutely necessary. The purpose of the operation, of course, is to remove diseased bone and inflammatory tissue, at the same time converting the mastoid antrum, epitympanum, middle ear, and auditory canal into one cavity accessible to observation and aftercare; thus, the causative pathology is eliminated and intracranial extension is prevented.

SUMMARY AND CONCLUSIONS

Acute and chronic otitis media including otitis media with effusion have been discussed in the light of modern thinking and trends in therapy. Certain pitfalls incident to antibiotic treatment of the acute ear have been noted, and the need for myringotomy has been emphasized. In the surgical management of chronic otitic suppuration, the modern, more direct approach by endaural mastoidectomy offers greater safety and shorter patient convalescence.

934 Republic Building.

The Pathology of Ectopic Pregnancy

Fred A. Brandt

Sophomore Student, University of Kansas School of Medicine

I should like to begin this paper with a definition of the term "ectopic pregnancy." In popular usage, this term has become almost synonymous with tubal pregnancy. However, in this paper I shall consider any implantation of a fertilized ovum occurring outside of the uterine cavity as constituting an ectopic pregnancy, a tubal pregnancy being but one type of ectopic pregnancy.

There are several types of ectopic pregnancy, each of which shall be discussed in turn. The types are tubal pregnancy, interstitial pregnancy, intraligamentous pregnancy, ovarian pregnancy, abdominal pregnancy, cervical pregnancy, and pregnancy in a rudimentary uterine horn.

Jarcho,[7] in his analysis of 1,225 cases of ectopic pregnancy of all types, gives the following incidences of the various types in his group of cases: the ovum implanted in the ampulla in 578 cases; the isthmus in 265 cases; the fimbriated extremity in 71 cases; the interstitial portion in 45 cases; the infundibula in 31 cases; the entire tube, including a hematosalpinx that contained products of conception, in 17 cases; the abdomen in 17 cases; the distal half of the tube in 10 cases; the distal two-thirds of the tube in 6 cases; the broad ligament in 5 cases; the entire tube and ovary in 5 cases; the cornu in 5 cases; tubo-ovarian in 2 cases; in a rudimentary horn in 1 case. In 164 cases, the site was either not stated or not proved.

Ectopic pregnancy occurs in approximately one of every 300 pregnancies and makes up approximately 1 to 2.5 per cent of all gynecological hospital admissions. It is most common in pregnancies associated with pelvic abnormalities, and it is more

common in Negroes than in whites, the ratio being 1 in 190 admissions to the hospital for whites and 1 in 130 admissions for Negros in one large group.[21] In this same group, the mortality for whites was .7 per cent and for Negroes, 3.9 per cent.

The causes of ectopic pregnancy are variable, but they may be classified under two main groupings. These are: conditions which prevent or retard the passage of the fertilized ovum into the uterine cavity and conditions which increase the receptivity of an ectopic implantation site to the fertilized ovum. Under the first of these classifications may be listed chronic salpingitis, by causing fusions of the folds of the tubal mucosa with narrowing of the tubal lumen or the formation of blind pouches; congenital and postembryonic developmental abnormalities of the tube, particularly diverticula; accessory ostia and hypoplasia; peritubal adhesions following postabortal or puerperal infection with kinking of the tube and narrowing of the lumen; tumors compressing the tube; previous surgery of the pelvis, particularly with resultant adhesions; and external migration of the ovum with resultant growth of the trophoblastic layer before the uterine cavity is reached. The second classification is predominantly concerned with ectopic endometrial tissue foci, particularly in the tubal mucosa.

I shall now proceed to the mechanisms of and the pathological lesions seen in the different types of ectopic pregnancy. The most common of these types, and therefore the most important to the clinician, is the tubal pregnancy, which I shall consider first.

As the uterus and tubes are genetically from the same origin and composed of the same tissues, the reaction to implantation of the fertilized ovum might be expected to be the same in both these tissues. Physiologically, this is true; however, in the tube the results are pathological from the onset, the organ being anatomically entirely unsuited for implantation or development of the ovum.

Until the time that the ovum begins to burrow into the mucosa, the process of development is identical with that found in normal pregnancy, but after this time, in tubal pregnancy, the conditions encountered by the ovum are entirely different. In the tube, there is little connective tissue stroma and no true decidua forms, although a "decidual reaction," with decidual cells throughout the mucosa, singly or in groups, does occur. At the base of the implantation site, no decidua basalis forms, or the little that does form is rapidly destroyed by the lytic action of the trophoblastic layer. The ovum, after penetrating the mucosa, comes immediately into contact with the inner circular muscular layer of the tube, and by its continuing lytic action, in search of an adequate blood supply and point of attachment, lyses the muscle fibers to such an extent that the tubal wall is irreparably weakened. The vessels eroded are of much larger size than those of the uterine decidua, resulting in hemorrhage rather than a normal blood supply into the intervillous spaces in the process of tubal implantation. This hemorrhage is often so extensive that the villi are disrupted and torn, the ovum capsule is distended and eventually ruptures, with resultant hemorrhage into the tubal lumen—the so-called "tubal abortion."

No decidua capsularis analogous to the uterine form develops, although decidual cells and groups of cells are often found. Mucosa and tubular folds can be demonstrated in the walls of the ovum sac nearest the tubal lumen, however, proving its "capsular" nature. This capsule is extremely weak, does not expand and grow with the ovum like true decidua, and, being constantly eroded by villi, ruptures early and easily.

One rather peculiar finding is the fact that the decidual reaction of the tubal mucosa is more extensive the further one progresses in his examination of the tissues distal to the site of implantation. This is seen in the fimbria, in the peritoneum covering the tube, and has even been observed in the tube opposite to the side of implantation. Thickening of the muscularis is seen at the site of implantation, and, at times, away from the implantation site, although generally the muscularis is modified in these locations only when stretched by the developing ovum.

In describing the termination of ectopic gestation in the tube, the terms "tubal abortion" or "tubal rupture" are used. That these terms are not strictly analogous to uterine abortion is apparent when it is realized that separation of the ovum from its attachment and expulsion by the muscular contractions of the tube do not occur. Although separation may occur and the ovum may be found free in the lumen of the tube, this is regarded as a passive carrying of the ovum into the tube by the pressure changes coincidental with rupture of the capsule, rather than the result of any active process initiated by the tubal musculature. That the ovum is often found protruding through the ostium is not regarded as a result of active expulsion by the tube, but rather as a result of implantation near the fimbriated end of the tube, development and enlargement of the ovum subsequently pushing the structure through the ostium following passive dilatation of the tube toward the fimbriated end.

If compression of the ovum does not result in pathological changes so extensive as to be incompatible with further growth, the extruded ovum may continue to grow, retaining its original tubal implantation site with eventual placental extension over the surface of the broad ligament and other nearby pelvic structures, forming the basis for most

abdominal pregnancies. Most authors believe that the ovum with its containing sacs must remain intact if further development is to occur. However, cases have been reported where only the remnants of the sac are present in the abdomen, usually forming adhesions, with the embryo or fetus completely free in the abdominal cavity.

If implantation occurs nearer the uterine end of the tube, termination will occur by any of three mechanisms—by rupture through the weakened tubular wall into the abdominal cavity; by rupture of the capsule into the tubal lumen; or, rarely, by separation of the ovum and its death, with subsequent hydatiform mole or polyp formation possible. The ovum may be pushed through the fimbriated end of the tube by the hemorrhage that occurs with the rupture, but, again, this is not due to any active participation in the process by the tubal musculature. This hemorrhage, in contradistinction to the mechanism in uterine abortion, is not always accompanied by separation of the ovum, but is due to the rupture of the ovum capsule alone. Rupture into the abdominal cavity may also occur secondary to the erosion of a large vessel by villi, the laceration produced being quite small. However, this may terminate fatally due to the constant blood loss occurring through this laceration.

One factor in tubal implantation that is often unrecognized is the effect occasioned by the physical site of implantation, whether on the "tip" of a mucosal fold, or in the peripheral portion of the tubular lumen in a depression between two folds. The latter is by far the most common site. In the former type, after invasion of the mucosa, the ovum lies in the interior of a fold of mucosa and is surrounded on all sides except at its base by this mucosa, greatly limiting its space for expansion. The latter type, as illustrated before, immediately after penetration of the mucosa, lies directly over the muscular layer of the tube with no lateral limitations to its space for expansion. For this reason, the "tip" implantation rarely remains viable and, if it does, the weakening of the muscular wall of the tube occurs later, with its subsequent rupture, than it does following the implantation in the depression between tubular folds.

During the course of tubal pregnancy, particularly if the ovum continues to develop for three months before termination, the uterus proper undergoes hypertrophy, and its endometrium is converted to a decidua vera by the action of the luteal hormones of the ovary, differing from the reaction seen in the normal pregnant uterus only in less marked development of the spongy layer and a greater abundance of blood spaces just beneath the free surface of the decidua. At termination, the decidua becomes degenerate and usually breaks away in small pieces. However, it may come off intact and represent a decidual cast of the uterine cavity. The vaginal "spotting" seen in many cases of ectopic pregnancy is thought to be due to early degenerative changes in this uterine decidua, involution resulting from constriction of the coiled arteries and sometimes reducing the thickness of the decidua by 50 per cent before massive sloughing occurs.

In interstitial ectopic pregnancy, the sequence of events with regard to the ovum is identical with those seen in tubal ectopic pregnancy. However, due to hypertrophy of the adjacent uterine horn musculature and the subsequent stretching similar to that seen in normal intrauterine pregnancy, the ovum may develop for weeks or even months before rupture occurs. When this rupture does occur, the resulting condition is similar to that seen with a ruptured uterus, severe hemorrhage and shock with death within one hour being a typical picture.

In a small number of cases, when the original site of implantation of the ovum is basiotropic, as described by Lichtenstein, that is, in the portion of the tube where the surface is not covered by peritoneum (within the broad ligament), rupture results in the contents of the gestational sac being extruded into the space formed by the separation of these folds of the broad ligament. This is the most "favorable" variety of rupture and terminates either by death of the ovum and the formation of a broad ligament hematoma or by a further development of pregnancy similar to that seen in "tubal abortion" and known as intraligamentous pregnancy. The favorable prognosis following this type of rupture is due to the hemorrhage being confined by the ligamentous folds and thus, being limited, with eventual resorption of the hematoma.

Ovarian pregnancy, as defined by the four criteria of Spiegelberg, demands that the tube, including the fimbriated end, be intact and distinctly separate from the ovary, that the gestational sac occupy the normal position of the ovary, that the sac be connected with the uterus by the utero-ovarian ligament, and that ovarian tissue be demonstrable in the walls of the sac. Many of the ovarian pregnancies reported in the literature do not conform to these criteria. When the sac is composed partially of tubal and partially of ovarian tissue, the term "tubo-ovarian pregnancy" is employed. These occur only with development of the ovum in a tubo-ovarian cyst, or in a tube whose fimbriated end is adherent to the ovary at the time of fertilization.

Various types of true ovarian pregnancy are recognized. These include the primary ovarian pregnancy in which ovarian tissue forms a complete intact layer around the ovum and its sacs. If rupture occurs, it must rupture through a previously intact layer of ovarian tissue, whether adherent organs or tissues covering this ovarian tissue are ruptured or not. Pri-

mary ovarian pregnancy is the result of fertilization of the ovum in situ in the Graafian follicle after rupture, implantation of the fertilized ovum in the Graafian follicle, or implantation in ovarian tissue other than the Graafian follicle. The other main type is the combined ovarian pregnancy, illustrated by the tubo-ovarian pregnancy previously discussed.

Termination of ovarian pregnancy follows the same pattern as seen in tubal pregnancy: rupture of the capsule followed by death of the ovum, usually accompanied by extensive hemorrhage into the abdominal cavity, or continued viability of the ovum with extension of the placenta over adjacent pelvic structures and the development of a secondary abdominal pregnancy. The changes in the uterus are identical with those seen in tubal pregnancy.

Abdominal pregnancy, in the majority of cases, is secondary to the rupture of the aforementioned types of ectopic pregnancy, with continued viability of the ovum. Usually the ovum, after extrusion into the abdominal cavity, becomes encapsulated and degenerates. Mummification, maceration, or calcareous infiltration (lithopedion formation) may then occur. However, a few cases are reported in the literature that are apparently examples of primary implantation of a fertilized ovum in the abdominal cavity. This condition is thought to occur primarily as a result of external migration of the ovum, with the formation of the trophoblastic layer while the ovum is within the abdominal cavity. It is accompanied by normal tubes and ovaries with no evidence of recent or remote injury to the tubes, the absence of any evidence of a uteroperitoneal fistula, and the implantation on a peritoneal surface, with the ovum young enough to eliminate the possibility of secondary implantation following a primary implantation in the tube.

One of the best examples of this type of implantation was described by Studdiford.[25] A rare form of primary abdominal implantation has been described where the fertilized ovum escaped from the uterine cavity through a defect in the uterine wall caused by the postoperative separation of the uterine wound following a previous cesarean section. It has been demonstrated that the peritoneum undergoes a "decidua-like" reaction in abdominal implantation, similar to that seen in tubal pregnancy. However, some authorities do not accept this finding, others believe that abdominal implantation occurs only at sites of ectopic "endometrial" tissue, while still others do not accept the evidence of ectopic "endometrial" tissue on a peritoneal surface. The entire question is controversial, as seen in a review of the literature.

Cervical pregnancy is regarded by many authors as a form of abortion rather than a true ectopic pregnancy. However, in my definition of ectopic pregnancy, this type must be included. It is characterized by ectopic foci of endometrial tissue in the cervical wall mucosa and is included under the increased receptivity of an ectopic implantation site grouping. As the mucosa of the cervix reacts poorly to the invasive action of the trophoblastic layer of the ovum, it is readily destroyed, and the developing placenta becomes anchored to the muscularis. This form of ectopic pregnancy rarely progresses beyond the 20th week of gestation and is usually terminated surgically because of bleeding.

A rudimentary uterine horn is the result of initial lack of development during fetal life of the middle and lower parts of one of the Müllerian ducts, with resulting failure of the two ducts to coalesce. A rudimentary cornu on one side, attached by a band of fibro-muscular tissue to the lower or middle part of the normal uterus of the other side, is the consequence. The tube and the ovary of the defective side are usually well developed, but only about 20 per cent show communication through a minute canal with the uterus.

Pregnancy in a rudimentary horn develops in one of three ways: fertilization through the minute canal, by transperitoneal migration of a spermatozoön, or by transperitoneal migration of the fertilized ovum. That any of these mechanisms may occur is shown by the location of the corpus luteum in the cases presented in the literature, this structure appearing in either ovary. About 90 per cent of these pregnancies terminate with rupture similar to that seen in interstitial pregnancy, the other 10 per cent continuing to term as a result of uterine tissue hypertrophy in the cornu, although the fetus is almost invariably dead "in utero." I find only one case in the literature where the child survived for more than six hours after delivery, this case showing the child alive and well at 2½ years.[15] This type of ectopic pregnancy shows essentially the same symptoms as an abdominal pregnancy, except that the fetus is enclosed in uterine tissue of anomalous origin within the abdominal cavity.

BIBLIOGRAPHY

1. Lull, Clifford B., and Kimbrough, Robert A.: Clinical obstetrics, J. B. Lippincott Co., Philadelphia, 15:420-422, 1953.
2. Titus, Paul: Management of Obstetric Difficulties, 3rd Ed., C. V. Mosby Co., St. Louis, 1945, XI, 305-335.
3. Eastman, Nicholson J.: Williams Obstetrics, 10th Ed., Appleton-Century-Crofts, Inc., N. Y. 1950, XXI, 501-528.
4. Potter, Edith L.: Pathology of the Fetus and the Newborn, Year Book Publishers, Inc., Chicago, 1952, III, 15-20.
5. Novak, Emil: Gynecological and Obstetrical Pathology with Clinical and Endocrine Relationships, 2nd Ed., W. B. Saunders Co., Philadelphia and London, XXX, 437-458.
6. Baden, W. F., and Heins, O. H.: Ovarian pregnancy, Amer. J. Obstet. Gynec. 64:353-358, 1952.
7. Jarcho, J.: Ectopic pregnancy with special reference to abdominal pregnancy, Amer. J. Surg. 77:273-313, 1949.
8. Deweese, W. J.: Ovarian pregnancy, Minn. Med. 32:272-274, 1949.
9. Beacham, W. D.; Collins, C. G.; Perry, J. E., and Beacham, D. W.: Ectopic pregnancy, JAMA 136:365-371, 1948.
10. Whitacre, F. E., and Lynn, H. D.: Early and Late Ectopic Pregnancy, Jour. Mich. St. Med. Soc. 47:285-288, 1948.
11. Branscomb, L.: Advanced abdominal pregnancy, Amer. Jour. Obstet. Gynec. 54:874-878, 1947.
12. Fraser, I. T.: Full-term intraligamentary pregnancy, Proc. Royal Soc. Med., London 40:378-379, 1947.

13. Thornton, M. J.: Ectopic pregnancy: An analysis of 67 consecutive cases, Wisc. Med. Jour. 46:691-693, 1947.
14. Isbell, N. P., and Bacon, W. B.: Primary ovarian pregnancy, Amer. Jour. Obstet. Gynec. 54:329-335, 1947.
15. Bate: Abdominal type of ectopic pregnancy, Amer. Jour. Surg. 72:258-261, 1946.
16. Marchetti, A. A.; Kuder, K., and Kuder, A.: Clinical evaluation of ectopic pregnancy, Amer. Jour. Obstet. Gynec. 52:544-555, 1946.
17. Leff, B., and Wrison, S. G.: Analysis of 212 consecutive cases of ectopic pregnancy, Amer. Jour. Obstet. Gynec. 53:296-299, 1947.
18. Burkons, H. F., Abdominal pregnancy, Ohio St. Med. Jour. 43:161-164, 1947.
19. Beacham, W. D.; Collins, C. G.; Beacham, D. W.; Griffon, J. P., and Nix, F. T.: Ectopic pregnancy, Jour. Missouri Med. Assoc. 48:629-633, 1951.
20. Scholtz, M.: Full-time pregnancy in a rudimentary horn of the uterus, Jour. Obstet. Gynec. British Empire 58:293-296, 1951.
21. Anderson, G. W.: Racial incidence and mortality of ectopic pregnancy, Amer. Jour. Obstet. Gynec. 61:312-320, 1951.
22. Collins, C. G.; Beacham, W. D., and Beacham, D. W.: Ectopic pregnancy, mortality and morbidity factors, Amer. Jour. Obstet. Gynec. 57:1144-1154, 1949.
23. Romney, S. L.; Hertig, A. T., and Reid, D. E.: Endometria associated with ectopic pregnancy. A study of 115 cases, Surg. Gynec. Obstet. 91:605-611, 1950.
24. Litzenberg, J. C.: Microscopical studies of tubal pregnancy, Amer. Jour. Obstet. Gynec. 1:223, 1920.
25. Studdiford, W. E.: Primary peritoneal pregnancy, Amer. Jour. Obstet. Gynec. 44:487-491, 1942.

Weber-Christian Disease: A Case Report

Charles Pokorny, M.D., and C. Alexander Hellwig, M.D.

Halstead, Kansas

Weber-Christian disease, or relapsing, febrile, nodular, nonsuppurative panniculitis, is characterized by recurring crops of inflammatory and necrotic nodules in the panniculus adiposus and is usually accompanied by a febrile reaction. The nodules may be single or multiple, tender or painless, freely movable and from 0.5 cm. to several centimeters in diameter. The lesions as a rule do not suppurate but may become necrotic and liquefy. The lesions most commonly occur on the legs, thighs, arms, or breasts, but may occur any place over the body.

Although the lesions may heal without residuals, usually there is a dimpling due to the fibrotic scarring of the subcutaneous fat. The relapses may be as short as a few weeks or as long as several years.

The disease was originally described in 1891 by Pfeifer. Weber, in 1925, described a case using the term "relapsing nonsuppurative panniculitis," and Christian,[5] in 1928, added the term "febrile" to the name.

As it originally was thought to be a rather benign disease, little attention has been paid to it, and for that reason it probably now passes undiagnosed many times. From the literature, Beerman[2] was able to collect 14 cases with a fatal termination. One was that of Mostofi and Engleman,[12] a 39-year-old Filipino male soldier who, at postmortem, showed lesions involving the adipose tissue of the skin, epicardium, peripancreatic, perirenal, and mesenteric areas.

Shuman[13] has pointed out that there are certain similarities in the histopathology to Whipple's disease, erythema nodosum, adiposis dolorosa, insulin granulomata, and scleroderma. While the lesions occur most frequently in the panniculus, they also occur in adipose tissue elsewhere.

Hallahan and Klein,[9] in their review of the literature, felt that although the etiology of relapsing febrile, nodular, nonsuppurating panniculitis was unknown, many factors were to be considered. The halogen group, especially the iodine and bromine compounds, could produce these lesions; also subcutaneous injections of camphor in oil, insulin, and paraffin would produce this response. No organisms nor filterable viruses could be consistently demonstrated from these lesions. Focal infections, septicemia, tuberculosis, renal failure, dietary deficiency, trauma, impaired fat metabolism, foreign body reaction, and endocrine disturbances have all been associated with but not proved as the causative mechanism of this disease. Friedenberg[8] felt Weber-Christian disease represented an allergic reaction to a variety of noxious stimuli.

The disease has occurred in about every age group, with the youngest a 6-month-old infant female, the oldest a 70-year-old female. Beerman states it is most commonly found in obese women between the ages of 20 to 40. However, the disease in recent years is reported with more equal distribution between males and females, although it had been thought to be more common in the female in the proportion of three to one. The disease has been reported in individuals from a number of countries, namely, the United States, Canada, Hawaii, China, Spain, Germany, Scandinavia, Syria, Britain, South America, the Philippines, and Australia. Johnson and Plice[10] reported a case in a Negro.

The onset of the disease is insidious and without prodromata. The nodules may occur with fever, chills, nausea, and malaise. The lesions may remain

From the Department of Internal Medicine, Hertzler Clinic and Halstead Hospital, Halstead, Kansas.

only a short time or for months. Any of the above symptoms may be absent, either singly or in combinations. The constitutional symptoms may be mild, neither incapacitating the individual nor requiring that he be hospitalized.

Beerman states the differential diagnosis must be made from: 1. subcutaneous fat atrophy, 2. insulin lipodystrophy, 3. hypodermic injections and drug addiction, 4. trauma, 5. erythema nodosum, 6. erythema induration, 7. sarcoid of Darier-Roussey, 8. dermatomyositis, 9. morphea, 10. adiposis dolorosa, 11. subcutaneous fat necrosis in the newborn, 12. benign nodular hypodermite, 13. thrombophlebitis, 14. nodular syphilis, 15. leprosy, 16. nodular vasculitis, 17. spontaneous panniculitis of Rothmann-Makai, 18. metastatic neoplasm and lymphoma. The clinical picture as well as the report of biopsy will give the correct diagnosis.

There is no specific therapy for this disease. The sulfonamides have been reported to give good results. Neither penicillin nor aureomycin has had any effect. Shuman has reported a two months' remission with x-ray therapy; in his case there was no response to antimicrobial agents, and cortisone therapy succeeded in producing a normal temperature for 5 days. Cortisone and ACTH brought about marked remission as reported by Drew et al.[6]

Symptomatic and supportive therapy seems best for the patient, although recently salicylates and rather heavy doses of antihistamines have been reported with equivocal results. Arnold[1] has reported good results from intermittent treatment with sulfapyridine. Friedenberg administered cortisone to one of his patients but had to discontinue it because the biopsy site broke down.

CASE REPORT*

A 57-year-old white female came to the clinic on February 19, 1953. Her presenting complaints were painful lumps or nodules which had been recurring for the past eight years. While she had never had any known fever when these nodes appeared, there was associated pain in the groins, also leg ache, backache, and at times nausea. If she remained on her feet very much, there was swelling of the feet.

She believed the nodules were recurring more frequently. When the nodules disappeared, a sunken area would remain. When the nodules first appeared, in 1945, they recurred at varying intervals; during the past year they had been following one another almost continuously, and she was almost completely disabled. It was her impression that trauma was the inciting factor.

Family history revealed that her father died at the age of 86 of cancer of the prostate. A paternal aunt had diabetes. She had had the usual childhood diseases with a history of typhoid fever and questionable rheumatic fever. She was married, with three living children. She had had a hysterectomy at the age of 47.

Physical examination revealed a rather obese white female of stated age. The blood pressure was 116/65, pulse 86 and the temperature 99° on admission, which was the highest during this observation. On the lateral surface of the left thigh was an indurated area about 5 cm. in diameter, which was tender and reddened. This seemed to be attached to the skin. Scattered over the lower extremities were dimpled areas which the patient stated were sites of former lesions.

Laboratory reports revealed the hemoglobin to be 82 per cent or 14 gm., red blood cells 4,250,000, white blood count 7,950, with 2 eosinophiles, 2 stabs, 38 segments, 55 lymphocytes, and 3 monocytes in the differential. The Kline was negative. The nonprotein nitrogen was 29 mg. per cent, creatinine 1.67 mg. per cent and urea nitrogen 12.5 mg. per cent. The sedimentation rate was 11 mm. per hour, by the Westergren method. The urine was negative for albumin and sugar, with a specific gravity of 1.016. Skin tests for histoplasmosis and coccidioidosis and the first and second tuberculin tests were negative. The chest x-ray was considered negative.

Biopsy on February 21, 1953, revealed tissue from the left thigh weighing 55 grams and consisting of skin of elliptical form measuring 6 x 3 cm., with a 3.5 cm. thick fat tissue attached. Multiple sections showed fat tissue arranged in lobules. Beneath the surface was a firm area, band-like, with the appearance of fat tissue with whitish, smaller nodules. This hard area, extending throughout the specimen, was 8 mm. thick. Microscopic findings showed the epidermis well preserved with normal structure. The corium showed collagenous fibers and groups of sweat glands; there were several blood vessels surrounded with lymphocytes, especially monocytes. The lesion was situated in the subcutaneous fat tissue and consisted of cellular accumulations of histiocytes. There were many small spaces which were surrounded with macrophages and giant cells. Many large cells with foamy cytoplasm were noted. There was no evidence of neoplastic growth. The pathological diagnosis was nonsuppurative panniculitis (Weber-Christian disease).

The patient recently reported she had had two mild episodes shortly after hospitalization. For about 5 months she had no recurrence, but in the latter part of September, 1953, a new area appeared with typical manifestations.

* The patient was referred by L. G. Heins, M.D., Abilene, Kansas.

HISTOLOGY

The microscopic changes in Weber-Christian disease are so similar to those seen in localized lipogranuloma of traumatic origin that neither lesion can be differentiated on histological grounds alone. In the earliest stage, lipogranuloma consists of alteration of fat cells in the subcutaneous tissue. By disruption of the cell membranes, fat droplets are liberated from the fat cells and form globules of considerable size. These globules become surrounded by macrophages and by giant cells of the foreign body type. Macrophages, distended with fat droplets, are seen throughout the affected tissue. They transport the fat debris into perivascular and perineural lymphatic vessels, and some reach regional lymph nodes. The swelling of the regional lymph nodes may lead to a wrong clinical diagnosis of Hodgkin's disease or cancer with metastases. Focal lymphocytic infiltration is usually present, but polymorphonuclear leucocytes, plasma cells, and eosinophilic cells are either rare or absent. Globules of masses of free fat finally become entrapped by fibroblasts, which form connective tissue and later become rather dense hyaline scar tissue.

ETIOLOGY

Lecene and Moulonguet[11] believe that the primary and basic factor preceding lipogranuloma is the transformation of neutral fat into irritating fatty acids and soaps by lipase. Lipase, according to these authors, can be liberated from blood following traumatic hemorrhage or can be carried to the injured area by the blood stream. Since blood pigment is usually absent in the lesions of Weber-Christian disease, and since autopsy studies have revealed, in addition to the subcutaneous lesions, the same changes in epicardial, peripancreatic, periadrenal, peri-anal, and mesenteric fat tissue, we must assume that in Weber-Christian disease the etiological agent is blood-borne and acts on the traumatized fat cells of the subcutaneous tissue.

The lipase content of the blood is dependent on the function of the pancreas. In contrast to the normal levels of serum amylase, in chronic disease of the pancreas serum lipase is increased in about one-half of the cases of carcinoma of the pancreas, in more than one-half of the cases of carcinoma of the papilla of Vater, in 10 to 15 per cent of the cases of gallbladder disease, occasionally in duodenal ulcer, hepatitis, liver cirrhosis, chronic pancreatitis, and pancreatic obstruction. In conditions not primarily involving the pancreas, increase in serum lipase has also been observed, indicating a secondary pancreatic involvement.

There is a single histological description of the pancreas in the literature on cases of Weber-Christian disease. Mostofi and Engleman found, in their fatal case of Weber-Christian disease, that the acinar epithelium of the pancreas had a pale vacuolated cytoplasm suggesting fatty or hydropic degeneration. We therefore propose, as a working hypothesis, an increase in serum lipase as one of the underlying causes of Weber-Christian disease. The released and activated lipase acts secondarily on traumatized fat tissue.

Our patient associated trauma with her disease. This has been reported by other authors.[2, 7] It is conceivable that on many occasions the trauma may be so slight as not to be remembered by the patient.

Bendel[3] has suggested that a noxious stimulus produces a metamorphosis of the subcutaneous fat. This in turn acts as a foreign body, producing a foreign body response, with the reticuloendothelial system playing a large part of the syndrome. It is thought that, if this were true, the trauma may so change

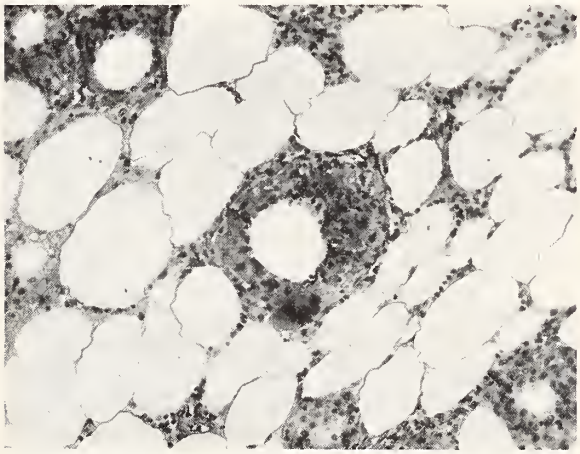


Figure 1. Early stage of lipogranulomatous lesion. In the center a ring of histiocytes and one giant cell surround a cystic space filled with altered fat.

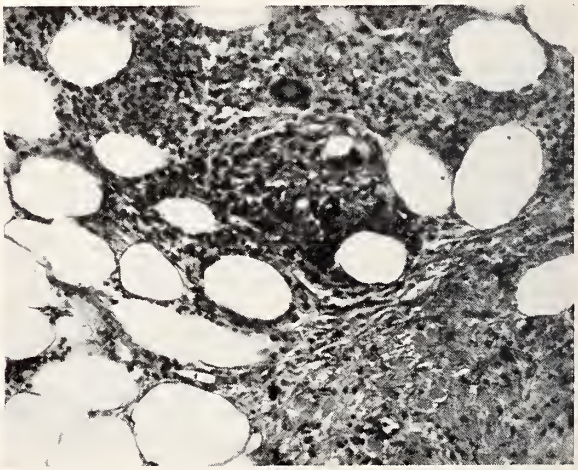


Figure 2. Later stage. Dense accumulation of lymphocytes and histiocytes with 2 giant cells. Fibroblasts have formed collagenous fibers.

the structure of the fat molecule as to bring about this response.

Allergic reasons have been suggested as causing the response.[3, 4, 6] Brudno[4] regarded the syndrome as an allergic systemic reaction to nonspecific stimuli with focal manifestations. He advances the theory that this is an antigen-antibody reaction. In our case no definite history of allergy could be determined.

CONCLUSIONS

Another case of Weber-Christian disease in which trauma is considered as an inciting factor is reported. In this case no history of febrile reaction could be obtained.

Elevation of serum lipase plus trauma may be the underlying cause of this disease.

BIBLIOGRAPHY

1. Arnold, H. L., Jr.: Relapsing febrile nodular nonsuppurative panniculitis (Weber-Christian disease); preliminary report of ease controlled by sulfapyridine, Arch. Dermat. & Syph. 51:94-99 (February) 1945.
2. Beerman, Herman: Weber-Christian syndrome with review of literature, Am. J. Med. Sc. 225:446-462 (April) 1953.
3. Bendel, W. L., Jr.: Relapsing febrile nodular panniculitis (Weber-Christian disease); review of the literature and report of ease, Arch. Dermat. & Syph. 60:570-596 (October) 1949.
4. Brudno, James C.: Chronic relapsing febrile nodular nonsuppurative panniculitis (Weber-Christian disease); relation to rheumatic fever and allied diseases, New Eng. J. Med. 234:513-517 (October 5) 1950.
5. Christian, H. A.: Relapsing febrile nodular nonsuppurative panniculitis, Arch. Int. Med. 42:338-351 (September) 1928.
6. Drew, D. W.; Wasson, A. A., and Morris, J. L. E.: ACTH and cortisone in the treatment of Weber-Christian disease; a ease report, J. Maine Med. Assoc. 42:357-359 (December) 1951.
7. Eisaman, J. L., and Schneider, L. A.: Nodular nonsuppurative panniculitis (Weber-Christian disease), J.A.M.A. 146:1417-1418 (August 11) 1951.
8. Friedenberg, Robert: Weber-Christian disease; report of two eases, Ann. Int. Med. 38:528-532 (Mareh) 1953.
9. Hallahan, J. D., and Klein, T.: Relapsing febrile nodular nonsuppurative panniculitis; review of literature and report of ease, Ann. Int. Med. 34:1179-1201 (May) 1951.
10. Johnson, W. A., and Pliee, S. G.: Relapsing febrile nodular nonsuppurative pannieulitis; report of ease in a Negro, with review of literature, Arch. Path. 48:281-286 (October) 1949.
11. Lecene, P., and Moulonguet, P.: Ann. d'anat. path. 2:193, 1925.
12. Mostofi, F. K., and Engleman, E.: Fatal relapsing febrile nonsuppurative panniculitis, Arch. Path. 43:417-426 (April) 1947.
13. Shuman, C. R.: Relapsing panniculitis; review of literature and report of case, including treatment with cortisone, Arch. Int. Med. 87:669-681 (May) 1951.

Addison's Disease Complicated by Pregnancy: A Case With Postpartum Hypertension

Thomas J. Luellen, M.D., and Ray A. West, M.D.

Wichita, Kansas

Pregnancy is a serious complication for the patient with Addison's disease, for it may precipitate acute adrenal cortical insufficiency. The first review of cases of Addison's disease with pregnancy reported a maternal mortality rate of approximately 50 per cent.[1] By 1950, accumulated case reports indicated that maternal mortality had progressively diminished by virtue of improved management of adrenal crisis. Since the use of cortisone, uneventful pregnancy despite Addison's disease has been reported,[2, 3] but available cases are too few for statistical analysis. It would appear, however, that effective management of pregnancy in Addison's disease depends upon anticipation of adrenal crisis and upon recognition of other potential complications which may occur during the pregnancy.

Adrenal crisis may initially appear during the first trimester of pregnancy, when hyperemesis, with electrolyte, water, and carbohydrate loss, is a precipitating factor. During the second and third trimesters, adrenal crisis is uncommon. Instead, desoxycorticosterone acetate overdosage may cause maternal death, as hormonal requirements are lowered by the sodium retaining effect of progesterone and the possible elaboration of adrenocortical-like substances by the placenta. Fetal adrenal secretion is probably insufficient to affect maternal electrolytes.[4] Maternal mortality in Addison's disease is threatened most frequently during delivery and puerperium, when hemorrhage, trauma, and withdrawal of the placental progesterone may precipitate adrenal crisis. The fetal mortality rate is insignificant among term pregnancies, and spontaneous abortion is uncommon. Therapeutic abortion is hazardous.

Postpartum hypertension is an event not usually anticipated as a complication in Addison's disease. In the case following, cortisone and desoxycorticosterone acetate were used in treatment, and the principal complication was hypertension occurring on the fifth and sixth postpartum days.

CASE REPORT

A white woman, age 20, gravida 1, para 0, was first seen during pregnancy at the Wichita Clinic

From the Departments of Medicine (Dr. Luellen) and Obstetrics-Gynecology (Dr. West), The Wichita Clinic, Wichita, Kansas.

MEDICAL LIBRARY - UNIVERSITY OF MARYLAND

on June 30, 1952, when she complained of nausea. The estimated date of confinement was February 9, 1953.

Past history revealed that in 1951 a dermatologist had recognized Addisonian pigmentation of one year's duration, and she visited the Mayo Clinic* soon afterwards, where the diagnosis was confirmed. Treatment had consisted of 5 mgm. of cortisone twice a day, 2 mgm. of desoxycorticosterone acetate twice a day buccally, and 3 gm. of supplementary salt daily. The blood pressure had been 96 to 106 mm. Hg. systolic, and 64 to 78 mm. Hg. diastolic.

Physical examination and routine laboratory examinations were normal, except for pigmentation of the lip and a generalized darkening of the skin. The weight was 127 pounds and the blood pressure 90/60. The blood chemistry determinations during and following the pregnancy are indicated in Table I.

TABLE NO. I
BLOOD DETERMINATIONS

Date	*Sodium Meq/L*	*Potassium Meq/L*	*Chlorides Meq/L*	*CO_2 Meq/L*	*Sugar Mgm. %*	*Urea Nitrogen Mgm. %*
July 3	139	3.95	100			
Sept. 3	142	4.55				
Jan. 8	147.5	5.00				
Jan. 16	137	5.40	106	10.6	80	
Jan. 17			104	12.8	81	11.2
Jan. 19	140		106	13.9		
Jan. 21	135.5	4.20	96	16.9		
Jan. 22	133	5.10	98	18.2		

During the first trimester, no hyperemesis occurred. Treatment included 10 to 25 mgm. of cortisone in divided daily doses, 2 mgm. of desoxycorticosterone acetate twice a day buccally, and 3 gm. supplementary salt daily. At three and one-half months' gestation, dependent edema appeared. Salt tablets and desoxycorticosterone acetate were voluntarily discontinued for three weeks, after which the blood pressure was 82/44 mm. Hg.

During the second and third trimesters, while the aforementioned treatment was continued, the blood pressure was 96 to 100 mm. Hg. systolic and 60 to 70 mm. Hg. diastolic. Several infections of the respiratory tract were tolerated without incident. Urinalysis throughout the course of pregnancy showed no albuminuria or abnormal cellular elements. No edema was present except for a slight amount for a few days each the 5th and 7th months of gestation.

On January 15, 1953, at 5:00 p. m. the patient was hospitalized, having had uterine contractions for 12 hours. Examination revealed slight vaginal bleeding; the cervical os was firm, uneffaced, and without dilatation. Fetal heart sounds became more distant because of increased uterine tonicity. A normal fetus was delivered at 1:35 a. m. on January 16 by cesarean section. A diagnosis of abruptio placentae was confirmed by inspection of the uterus and placenta.

On January 15, before hospitalization, the patient had taken 10 mgm. of cortisone orally. At 5:00 p. m., on January 15, she received 100 mgm. of cortisone intramuscularly and 25 mgm. orally, and the blood pressure was 104/70 mm. Hg. At 10:30 p. m., 25 mgm. of cortisone was given orally, and at 12:45 a. m., on January 16, 100 mgm. of cortisone was given intramuscularly prior to surgery. During surgery she received methoxamine hydrochloride (Vasoxyl R), and the maximum blood pressure was 140/90 mm. Hg.

At 8:00 a. m. on January 16, 50 mgm. of cortisone was given intramuscularly, and an injection of 1,000 cc. of 10 per cent glucose in physiologic saline was begun. This was the only occasion on which intravenous solution was given. Salt was added generously to the diet. On January 16, laboratory data was satisfactory except for indicating acidosis, for which 0.6 gm. of sodium bicarbonate three times a day was given. Unfortunately, the oral intake of water and salt was not measured, for the patient felt unusually well during the first five days postpartum. Urinary output and 24-hour urine chloride excretion were determined (Table II). Treatment and blood pressure levels are indicated in Figure I. The regular dose of 2 mgm. of desoxycorticosterone acetate two times a day was given on three of the first five postpartum days.

TABLE NO. II
URINE EXCRETION

Date	*24 Hour urine Volume in cc.*	*24 Hour Chlorides (Total meq.)*
Jan. 17	2200	66
Jan. 18	2600	143
Jan. 19	2600	98
Jan. 20	2300	161
Jan. 21	3000	240
Jan. 22	2500	225
Jan. 23	1900	238
Jan. 24	1700	187
Jan. 25	2500	225

On January 21 at 2:00 a. m., the fifth postpartum day, she complained of headache. The blood pressure was 170/100 mm. Hg. Physical examination showed no edema. Serum electrolyte determinations were satisfactory, and less acidosis was present. This incident occurred 40 hours after she received 50 mgm. of cortisone intramuscularly. Diuresis had occurred during the preceding days, reaching its peak on January 21.

On January 21, the dose of desoxycorticosterone acetate was decreased to 2 mgm. daily for the next

* This case is being included as one of a series by Dr. William M. McConahey and Dr. A. B. Hunt, Mayo Clinic, in another publication.

two days, and the administration of sodium bicarbonate was discontinued. The last injection of 50 mgm. of cortisone was given on January 21, while the regular dose of cortisone was continued.

On January 22, at 6:00 a. m. she again had a severe headache. The blood pressure was 190/100 mm. Hg., decreasing by 8:00 a. m. to 140/80 mm. Hg. without treatment. Thereafter, the blood pressure gradually decreased to the prenatal level, and the patient had no more headaches. She continued to receive the same treatment prescribed before pregnancy.

DISCUSSION

Although acute adrenal insufficiency is the most common severe complication of Addison's disease during late pregnancy and early puerperium, the development of a temporary hypertension has been reported several times. This hypertension has been either unexplained or attributed to overdosage of salt and DCA, or to late toxemia of pregnancy. Hypertension was attributed to overdosage of salt and DCA in separate cases reported by Brent,[5] Loeb,[6] and Richards.[7] Brent's case developed an elevation of blood pressure on the first postoperative day. Loeb had a case with hypertension during the final two months of pregnancy. Richards' case was treated with Eucortone, salt, and DCA, and had an elevation of blood pressure and edema after delivery requiring reduction of the DCA dose on the fourth postpartum day.

Cohen[8] reported a case of preeclamptic toxemia with edema, albuminuria, and a blood pressure of 188/128 during labor, with a normotensive state following postpartum diuresis. This was an unusual occurrence, since Brent found no reports of eclampsia among 40 cases which she reviewed.

Galloway[9] reported a case with acute adrenal insufficiency at 7 months gestation; an undesignated number of days postpartum, the blood pressure rose to 170/80. He attributed the hypertension to overactivity of the adrenal glands with the cessation of pregnancy.

One of the patients reported by Knowlton, Mudge, and Jailer[10] in 1949 had hypertension during the last month of pregnancy, but she was normotensive postpartum. In 1950, Jailer et al.[11] reported a case with a blood pressure of 160/100 on the sixth postpartum day.

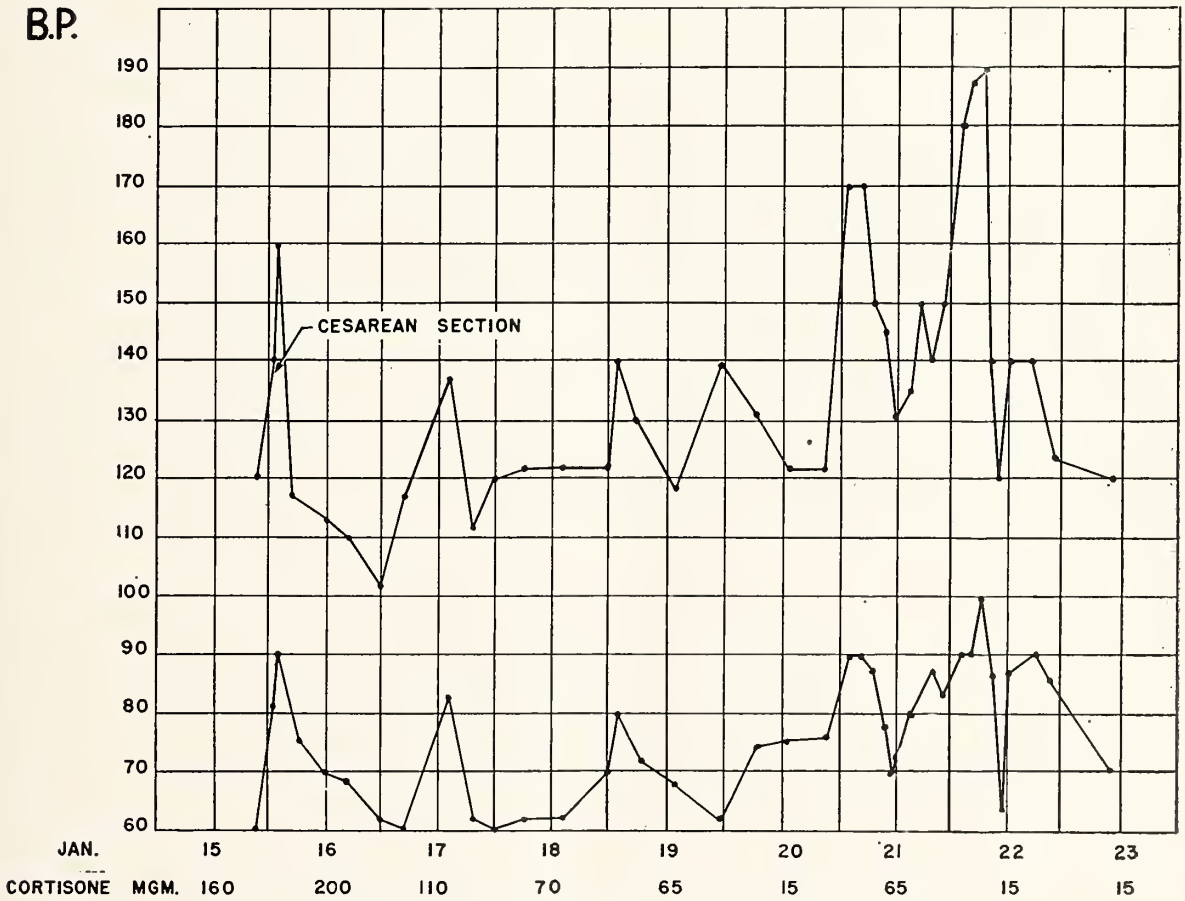


Figure I. The postpartum blood pressure and daily dosage of cortisone are shown.

In our case we lack evidence determining the cause of hypertension although several possible contributing factors may be considered. It is doubtful that the salt-retaining effect of the increased sex steroids during pregnancy[12, 13] was an important factor since the blood pressure was normal until after the time of delivery, there was a good urinary output during the entire early puerperium, and the patient's weight gain during the pregnancy was only 23 pounds. In the absence of edema and albuminuria, we believe toxemia was not present in this case.

There are several mechanisms by which cortisone might have influenced the blood pressure. Cortisone has been shown to increase the blood volume by shifting fluid from the intracellular to the extracellular[14] space. It also has the property of increasing blood pressure in Addison's disease or renal insufficiency,[16] in cases in which the weight gain and fluid retention are not considered sufficient to be the cause. Cortisone may have a vasoconstrictor effect since adrenalectomized patients will have a prompt rise in blood pressure when given cortisone with nor-epinephrine.[17] Also, cortisone decreases the hypotensive effect of tetraethylammonium chloride.[18]

However, we doubt that cortisone was the cause of the hypertension in our patient, since other cases mentioned in the literature had hypertension without large doses of cortisone; furthermore, the case reported by Wells and West received cortisone in an amount similar to ours without developing hypertension. Further information might have been obtained from our patient had we been able to repeat the same medication while she was not pregnant to establish whether hypertension might have been produced. The acidosis was thought to be due to the patient's Addison's disease, but renal insufficiency must be more thoroughly excluded.

We believe that there may be some as yet undetermined factor related to late pregnancy in the postpartum period in Addison's disease which predisposes to hypertension. More detailed studies of the electrolyte pattern and vascular responses during the postpartum period in Addison's disease seem to be indicated.

SUMMARY

1. A case of Addison's disease complicated by pregnancy, with survival of both the patient and baby, is reported.

2. The chief complication of postpartum hypertension is discussed.

BIBLIOGRAPHY

1. Fitz-Patrick, G.: Addison's disease complicating pregnancy, labor or the puerperium, Surg., Gynec. & Obst. 35:72-76 (July) 1922.

2. Wells, C. N., and Wesp, J. E.: Management of Addison's disease during pregnancy with cortisone and desoxycorticosterone, Am. J. Obst. & Gynec. 63:925-26 (April) 1952.

3. Rolland, C. F.; Matthews, J. D., and Matthew, G. D.: Pregnancy in Addison's disease, J. Obstet. & Gynaec., Brit. Emp., 60:57-60 (February) 1953.

4. Venning, E. H.: Adrenal function in pregnancy, Endocrinology 39:203-220 (September) 1946.

5. Brent, Florence: Addison's disease and pregnancy, Am. J. Surg. 79:645-52 (May) 1950.

6. Loeb: Cited by Cohen.[8]

7. Richards, T. A.: Addison's disease and pregnancy, Brit. M. J. 1:421 (February 23) 1952.

8. Cohen, M.: Addison's disease complicated by toxemia of pregnancy, Arch. Int. Med. 81:879-887 (June) 1948.

9. Galloway, C. E.; Sutton, D. C., and Ashworth, J.: An acute crisis of suprarenal insufficiency complicating pregnancy, Am. J. Obst. & Gynec. 40:148-49, 1940.

10. Knowlton, A. I.; Mudge, G. H., and Jailer, J. W.: Pregnancy in Addison's disease: Report of four patients, J. Clin. Endocrinol. 9:514-28, 1949.

11. Jailer, J. W., and Knowlton, A. I.: Simulated adrenocortical activity during pregnancy in an Addisonian patient, J. Clin. Investigation 29:1430-36 (November) 1950.

12. Samuels, L. F.; Evans, G. T., and McKelvey, J. L.: Ovarian and placental function in Addison's disease, Endocrinology 32:422, 1943.

13. Thorn, G. W., and Harrop, G. A.: Sodium-retaining effect of sex hormones, Science 86:40, 1937.

14. Dingman, J. F.; Streeten, D. H. P.; and Thorn, G. W., Cited by Thorn, G. W.; Jenkins, D.; Laidlaw, J. C.; Goetz, F. C.; Dingman, J. F.; Arons, W. L.; Streeten, D. H. P., and McCracken, B. H.: Pharmacological aspects of adrenocortical steroids and ACTH in man, New England J. Med. 248:284-94 (February 12) 1953.

15. Perera, G. A.; Pines, K. L.; Hamilton, H. B., and Vislocky, K.: Clinical and metabolic study of 11-dehydro-17 hydroxy-corticosterone acetate (Kendall Compound E) in hypertension, Addison's disease and diabetes mellitus, Am. J. Med. 7:56-69 (July) 1949.

16. Perera, G. A.: Cortisone and blood pressure, Proc. Soc. Exper. Biol. & Med. 76:583-85 (March) 1951.

17. Thorn, G. W.; Jenkins, D.; Laidlaw, J. C.; Goetz, F. C.; Dingman, J. F.; Arons, W. L.; Streeten, D. H. P., and McCracken, B. H.: Pharmacological aspects of adrenocortical steroids and ACTH in man, New England J. Med. 248:323-337 (February 19) 1953.

18. Brust, A. S.; Ransohoff, W., and Reiser, M. F.: Blood pressure responses to ACTH and cortisone in normotensive and hypertensive subjects in the resting state and during antonomic blockade with tetraethylammonium chloride, J. Clin. Investigation 30:630, 1951.

Have you ever rightly considered what the mere ability to read means? That it is the key which admits us to the whole world of thought and fancy and imagination? To the company of saint and sage, of the wisest and the wittiest at their wisest and wittiest moment? That it enables us to see with the keenest eyes, hear with the finest ears, and listen to the sweetest voices of all time? More than that, it annihilates time and space for us.—*James Russell Lowell.*

BLUE SHIELD

More About Enrollment

Plans for the 1954 Blue Cross-Blue Shield non-group community enrollment program were announced in the public press early this month, along with a list of the annual enrollment months for 99 of our counties. Activity is also going ahead for the continuous enrollment experiment which is to be conducted in Shawnee, Sedgwick, Osborne, and Barber counties.

While the opening dates for these four counties where there is to be continuous enrollment is still uncertain, campaign activities are already under way in many of the other counties, and some enrollments have already been held. In the new program, annual enrollment months have been established in every county included in the Kansas plan. Exact dates within each month may vary, but the month in which the non-group enrollment occurs will be the same every year. Enrollment months, except where there is continuous enrollment, are as follows:

January: Brown, Cheyenne, Coffey, Ellsworth, Nemaha, Osage, Rawlins, Rush, and Washington.

February: Cherokee, Cowley, Doniphan, Douglas, Lincoln, McPherson, Ottawa, Rooks, Saline, Sherman, Harvey, Miami, and Montgomery.

March: Bourbon, Ellis, Finney, Franklin, Jewell, Labette, Marion, Marshall, Pratt, Reno, Smith, and Thomas.

April: Chase, Chautauqua, Clay, Dickinson, Elk, Haskell, Gray, Jefferson, Lane, Linn, Seward, Meade, Morris, Republic, Rice, Russell, Scott, and Sumner.

May: Butler, Geary, Greeley, Hodgeman, Jackson, Lyon, Mitchell, Ness, Pawnee, Phillips, Stafford, Wallace, and Wichita.

June: Crawford, Greenwood, Harper, Kingman, Wilson, and Woodson.

September: Anderson, Barton, Graham, Riley, Sheridan, and Wabaunsee.

October: Allen, Clark, Comanche, Edwards, Ford, Kiowa, and Neosho.

November: Atchison, Cloud, Decatur, Grant, Hamilton, Kearny, Morton, Norton, Pottawatomie, Stanton, and Stevens.

December: Gove, Leavenworth, Logan, and Trego.

RETENTION IMPORTANT

It has already been pointed out that this new method of "regular" enrollment activity can be possible by the introduction of a new type of application which contains a fairly detailed and comprehensive statement of health. This new health statement-type of application card, however, will apply only to the *new* members (or applicants).

Here, again, the co-operation of Blue Shield participating physicians will be necessary in helping to remind all present members that they are not affected by this new program unless they drop their membership, and then seek to re-enroll. It cannot be emphasized too strongly to present members that they are still members in good standing, and that the valuable benefits of Blue Cross-Blue Shield are still theirs with no changes.

WAITING PERIODS

It has been necessary to establish a new group of conditions which are subject to an eight-month waiting period. They are as follows:

1. Obstetrical services.
2. Operations for the removal of tonsils and/or adenoids.
3. Treatment for tumors or growths.
4. Operations for hernia.
5. Operations for conditions of gall bladder, rectum, or genito-urinary tract.

While there may be some objection to this from the doctors, when one considers the alternative to establishing this clear-cut list of conditions, the latter course seems the best one to follow. Otherwise, it would be necessary to determine whether or not each case was for a condition which pre-existed the date of membership. This would involve considerable correspondence with doctors and a great deal of poor public relations and misunderstanding. On the other hand, the establishment of a clear-cut list on which there would be an eight-month waiting period will not involve all this administrative difficulty.

Blue Cross-Blue Shield will need the utmost in co-operation from the physicians and hospitals in Kansas. The underwriting of new memberships, the decision whether to refuse an applicant entirely or to accept him with a rider excluding treatment for certain conditions—these are situations in which the medical profession will be called upon to help the lay staff. No doubt it will be well worth some sacrifice of time and effort if it can result in a program of enrollment which can offer this coverage to every eligible person in Kansas.

The keystone of every public health endeavor must always be the private practitioner of medicine, who must rely on the local health department for assistance. If the health department is properly organized and carries out the objective for which it was designed, it will support and improve the private practice of medicine.—*Vlado A. Getting, M.D., J.A.M.A., Sept. 26, 1953.*

K.M.S. annual meeting, May 2-6, 1954, Topeka.

PRESIDENT'S PAGE

DEAR DOCTOR:

The 19th of February there will be an A.M.A. sponsored meeting in Denver on veterans' medical care. At this writing, it is the intent of your president to attend.

The thinking of the A.M.A. on veterans' medical care has always been in total accord with full and complete and continued medical care for service connected disabilities. But the A.M.A. has rigidly opposed medical care for non-service connected disabilities and the extension of the time of presumption of service connection to the point where it is becoming ridiculous.

It is certainly the duty of our government and us as taxpayers to give every veteran who was disabled by service connected injury or illness every consideration in the care of that disability, in order that he may return to a useful and happy life. But it is neither our duty nor our obligation on a national scale to take care of the medical needs of any veteran with an illness that has no service connection. This is pure socialization of medicine, and it has no place in the American way of living. It is just another wedge in the door that leads to the pathway to socialism.

Each and every one of us should give this matter our closest attention. We should aid the A.M.A. in its fight to curtail the further expansion of Veterans Administration hospital facilities and medical care for those conditions which should be procured from the facilities that exist in our local communities.



EDITORIAL COMMENT

Prehistoric Medicine

Skeletal and mummified remains of ancient man have afforded anthropologists a considerable, although by no means a uniform, opinion upon disease in prehistoric time. A pamphlet on this subject published by Ciba Pharmaceutical Products, Inc., some time ago was recently reviewed and appeared of sufficient interest to warrant a résumé at this time.

Arthritis and spondylitis appear among all people regardless of climate, diet, life habits, or period of time. A Neanderthal skeleton of about 40,000 B.C. is an example, and in the neolithic period some 30,000 years later numerous remains attest to the presence of vertebral ankylosis and atrophied intervertebral discs. The cave man from the Old Stone Age in Europe suffered from arthritis deformans, and the cave bears he fought had it also. One scientist reports that 40 per cent of the Egyptian skeletons showed spondylitis. In addition, many had arthritis of knee and shoulder joints as well as ankylosis of the pelvis.

Tuberculosis also seems to be identifiable in the remains of ancient man, although this condition is not so prevalent. It appears sporadically in skeletons from antiquity but is notably absent as, for example, in the 6,000 skeletons from Nubia dating about 3,500 B.C. Tuberculosis appears earlier in Europe than in Egypt. One Bronze Age tuberculous spine was found in France, and many evidences were obtained from the Aztec and Inca Indians.

Rickets appeared in early man, but its incidence was apparently not high. Some authorities insist evidences of this condition may be located as far back as the Cro-Magnon man. Rickets appear to have been more frequent among the Indians in this hemisphere than among European aborigines.

Numerous cranial deformities have been noted in the remains of ancient skeletons from all periods. Various authorities of presumed integrity have claimed to have identified "button osteomata" and leontiasis ossea in Chinese prehistoric crania.

Fractures were of course common in prehistoric man. An interesting feature is that forearm fractures in females among French neolithic skeletons are so frequent that they appear to be the rule rather than the exception. Head injuries were the most common of all traumatic lesions, probably inflicted through warfare and as the quickest method of ceremonial killing. Some authorities hold that the latter appears common enough to have been in wide practice. Evidence of healed injuries to the head are plentiful, and long before the era of written history trepanning became common in medical practice.

Prehistoric syphilis has been identified through skeletal diagnosis from many areas of the world, but principally in prehistoric Peru, Mexico, and North America. The criteria are found in cranial lenticular exostoses or lesions, premature closure of vault sutures, sabre-tibia and tibial hyperostosis, ogival form of the palate, and dental malformations, as erosion, impaction, microdontism, hypercementosis, bifid roots, Carabelli's cusp, and hypoplastic enamel.

A dissertation upon prehistoric man's knowledge of anatomy, pathology, and medicine becomes theoretical. It can be argued, however, that man's observations and instincts afforded him greater knowledge even in antiquity than may presently be realized. For example, certain primitive people today possess knowledge of anatomy and surgery that is amazing, and from the evidence of healed fractures it appears that some form of medical care must have been given. It must further be presumed that injuries to the skeletal structure must frequently have caused deep wounds which certainly would have required some form of closure. There is also evidence from early pictorial records that ancients in some parts of the world performed cesarean sections.

No knowledge is available as to when splints were first applied to fractures or when casts were used. It is, however, not unreasonable that the easing of pain through an immobilized limb would readily be recognized and repeated under similar circumstances. There are sufficient amputations to indicate that this also was frequently resorted to, possibly following a mangling injury or a poisonous bite.

The earliest known surgical treatise exists on papyrus from the 18th dynasty of Egypt. This describes the case history of 48 instances of trauma. In this work Egyptian surgery was at least advanced to the point where hemorrhage was arrested by pressure, where foreign bodies were extracted, where minor amputations were performed, wounds closed, dislocations reduced, abscesses cauterized, cysts incised and drained, and fatty tumors removed. In the treatment of wounds there was exploration, cleansing, and suturing or plastering of the wound edges. Described in this document were enemas, gargles, ear drops, and medicated pledgets in body cavities. There was no antisepsis, but there were ideas of fumigation. Obstetrics was in the hands of midwives, but there were specialists for the eye and for the belly. Descriptions of diseases from this period indicate the presence of "renal and vesicular calculi, arteriosclerosis, anthracosis, pneumonia which had progressed to hepatization, appendicitis, cirrhosis of the liver, splenic hypertrophy, genital prolapse, vesico-vaginal fistula . . ." and others including smallpox.

The physician of that period was versed in detail

concerning the evidences of certain conditions, the course of the disease, and the prognosis. It was recommended that upon seeing the patient he pronounce one of three verdicts: the first, "an ailment I can treat"; the second, "an ailment I will contend with"; and third, "an ailment not to be treated." The author of this article reminds his readers that this is faith, hope, failure.

Events in Washington

Events in Washington are transpiring with such rapidity that the lapse of time between the preparation of material and its appearance in the JOURNAL may render mention of the information obsolete. This, then, is historic as of the close of January, 1954.

The President has sent numerous messages to the Congress and has frequently mentioned items of interest to medicine. The following excerpts come from President Eisenhower's State of the Union message:

Socialization of Medicine: "I am flatly opposed to the socialization of medicine. The great need . . . can best be met by the initiative of private plans. But it is unfortunately a fact that medical costs are rising and already impose severe hardships on many families. The federal government can do many things and still avoid the socialization of medicine."

Research: "The federal government should encourage medical research in its battle with such diseases as cancer and heart ailments, and should continue to help states in health and rehabilitation."

Hill-Burton Program: "The present . . . act should be broadened to assist in the development of adequate facilities for the chronically ill . . . of diagnostic centers, rehabilitation facilities and nursing homes."

Reinsurance of Health Plans: "The war on disease . . . needs a better working relationship between government and private initiative. . . . A limited government reinsurance service would permit the private and non-profit insurance companies to offer broader protection to more of the many families which want and should have it."

Rehabilitation: "The program for rehabilitation of the disabled especially needs strengthening. . . .This program presently returns each year some 60,000 handicapped individuals to productive work. Far more disabled people can be saved each year . . . if this program is gradually increased."

Military Dependents: "Pay alone will not retain in the career service . . . the necessary numbers of long-term personnel. I strongly urge, therefore, a more generous use of other benefits important to service morale. Among these are more adequate living quarters, and medical care for dependents."

Medical Tax Deductions: " . . . we propose more liberal tax treatment for dependent children who work, for widows or widowers with dependent children, and for medical expenses."

Veterans: "The internal reorganization of the Veterans Administration is proceeding with my full approval. When completed, it will afford a single agency whose services, including medical facilities, will be better adapted to the needs of those 20 million veterans to whom the Nation owes so much." (There was no further reference to VA in the address.)

Social Security: "I ask that this extension (to 10,000,000 more persons) soon be accomplished. This and other major improvements . . . will bring substantial benefit increases and broaden the membership of the insurance system, thus diminishing the need for Federal grants-in-aid. . . ."

Since that time the President has spoken more completely upon the subject of Federal Health Service Reinsurance and on loans to non-profit health associations. He has further urged mandatory coverage for doctors and other self-employed and professional people under social security. These issues have not yet been clarified through the introduction of legislation so should perhaps not be discussed until details are more completely available.

Doctors at War and at Home

The total number of American service men wounded in World War II was about 700,000, of which the Army accounts for nearly 85 per cent. This figure is about three times as high as the first war.

American doctors drew high praise for their work since the fatality rate among those wounded was the the lowest in history for any army in the world. The fatality rate in the Army was 4.4 per cent, which is slightly more than half of the 8.3 per cent in the Army during World War I. Even a greater contrast was evidenced in the Navy and Marines where, in World War I, 12 per cent of those wounded died, as compared to 2.9 per cent in the second war. Figures available from the Korean fighting show the fatality rate among our wounded as 2 per cent, which of course is less than half percentagewise of the record breaking 4.4 per cent of World War II.

Much of this success is attributed to the use of sulfa drugs on a wide scale and the speeding up and development of penicillin. Use of blood and blood derivatives played an important role, as did effective methods of treating shock due to blood loss.

Many medical problems were met in World War II. One important factor was the problem of malaria control, when our major source of supply of quinine

was cut off by Japanese conquest of the East Indies. This problem was overcome by the spraying of disease-bearing mosquito breeding places and the use of new anti-malaria drugs which were developed and manufactured on a large scale.

Control of venereal disease, the vaccine for yellow fever, and the use of DDT were also aiding factors in the second war.

Much can also be said for the doctor population who remained at home to care for our civilian population. Doctors, overburdened with work, used resources so efficiently that instead of an expected increase in civilian mortality the opposite was quite apparent. During the years 1941 through 1945, the death rate in our country declined about 15 per cent, as compared to the preceding five-year period.

Where statistics are available in European countries, the opposite was true. The death rate among the civilian population in France showed an increase of 30 per cent in one year, the Netherlands 75 per cent, Belgium 20 per cent.

In summation, we find that while the doctors in service were instrumental in establishing new records in World War II, by cutting in half the fatality rate among the wounded, the civilian doctors held their own by aiding in the decrease of civilian mortality by over 15 per cent, as compared to the pre-war period.

Memorial Fund

The Woman's Auxiliary to the Kansas Medical Society has recently created a memorial fund which represents a departure from previous custom and a subject that well might occupy the attention of the medical society. The Auxiliary currently recommends that instead of bestowing flowers to the memory of deceased esteemed members, the Auxiliary will accept contributions to a nurse scholarship fund.

It is anticipated this will be used in the form of a loan that will be repayable according to terms defined by a special committee already appointed, and that interest on this loan will be low and possibly will begin after the student has completed her education. The present committee consists of Mrs. William Brewer, Hays; Mrs. Richard O'Donnell, Ellsworth, and Mrs. William T. Braun, Pittsburg.

Contributions to this special memorial fund may be given by any friend in the name of any Auxiliary member. Loans will be made through this fund by the committee according to applicants for loans and recommendations from the various schools of nursing in Kansas. In the beginning this must be used cautiously because the amount is small, but as this memorial fund increases in size it is hoped that many student nurses may benefit therefrom.

It is mentioned here as a worthwhile project on the part of the Auxiliary and because this represents an effort to which numerous doctors may wish at different times to contribute.

Vaccinia Immune Gamma Globulin

The Kansas State Board of Health calls attention to an attempt to gauge the effectiveness of a specially prepared vaccinia immune gamma globulin. This gamma globulin, manufactured by the Department of Pediatrics at the University of California Medical Center, is to be used in the prevention and therapy of serious complications of smallpox vaccination. Any physician who sees such complications is urged to contact the researchers immediately so that a supply of the material can be made available.

The complications, in the main, are generalized vaccinia, eczema vaccinatum, and progressive vaccinia (failure to produce specific antibodies) or vaccinia necrosum. Most patients so affected will be seen by physicians in clinical practice, in contagion hospitals, in teaching centers, public health departments, and well baby clinics.

Vaccinia immune gamma globulin is a solution of the globulin component of human blood collected from volunteer donors of the armed services who have been successfully vaccinated against smallpox from four to eight weeks prior to this blood donation. It was found that their neutralizing antibody titers were maximal at that time. The material was tested in India for the specific prevention of smallpox in exposed susceptible family contacts, and experience indicates that it is successful.

It is the thought of the University of California physicians that vaccinia immune gamma globulin can be distributed to best advantage on a request basis; the supply is not large enough to allow national distribution for storage.

Between 20 and 60 per cent of the cases of complications of smallpox vaccination are fatal, despite intensive fluid and antibiotic therapy. Therefore, it is essential that physicians seeing such patients communicate with the researchers by telephone or telegram so that the material may be dispatched by air express, free of charge. The address to be used is C. Henry Kempe, M.D., Department of Pediatrics, University of California Hospital, San Francisco 22, California, MOntrose 4-3600, Extension 371. The university hospital will refer night calls to laboratory personnel.

Make hotel reservations now for the annual meeting, May 2-6, 1954, Topeka.

ACTIVITIES OF MEMBERS

Dr. James P. Haigler, Hays, announces that Dr. Rex C. Belisle is now associated with him in general practice. Dr. Belisle, a graduate of the University of Kansas School of Medicine, practiced for a short while in Plainville before beginning a three-year tour of duty with the Navy.

Dr. Winstan L. Anderson, Atchison, has been appointed to a two-year term as a member of a new city-county health board. He will represent the city. Dr. Edwin T. Wulff, representing the county, has been appointed to a three-year term on the same board.

Dr. Lee Rook, Kansas City, took office last month as president of the Wyandotte Academy of General Practice. Other officers are: Dr. M. R. Fitzpatrick, vice-president; Dr. Harvey L. Lloyd, secretary; Dr. A. W. Bradford, treasurer. Members of the board of directors are Doctors Emmerich Schulte, Frank J. Strick, and Gaylord P. Neighbor.

Dr. John B. Dixon, Parsons, was licensed as a private pilot last month.

Dr. Paul H. Wedin, Wichita, was speaker at a recent meeting of the Dickinson County Medical Society. His subject was "Surgical Disease of the Chest."

Dr. Lynn C. Hartman, director of the Cowley County Joint Board of Health during the past three years, resigned that position last month to devote her time to her home and family. Dr. Roscoe F. Morton is serving as health director pending appointment of a permanent director.

Dr. Warren F. Bernstorf, Winfield, took part in a panel discussion on "Ministry to the Sick," during the Kansas Christian Ministers' Institute held at Winfield early in January.

Dr. John L. Fatland, Council Grove, spoke before the Rotary Club in that city last month on the subject of "Trends in Medical Care."

Dr. Joseph E. Gootee was recently elected president of the Topeka Blood Bank for the year 1954, and Dr. G. F. Helwig was named secretary. Serving on the board of directors are Doctors John W. Cavanaugh, John L. Lattimore, Howard U. Kennedy, and David E. Gray.

Dr. D. M. Diefendorf, Waterville, has been named health officer of Marshall County. He replaces the late Dr. R. L. McAllister.

Dr. A. K. Ratzlaff, Goessel, was speaker at a meeting of the Fourth District Nurses' Association at Halstead last month.

Dr. O. R. Cram, Larned, and Dr. Mary Glassen, Phillipsburg, were recently elected directors of the Kansas Association for Mental Health.

Dr. William C. Menninger, Topeka, addressed the 53rd annual meeting of the Family Consultation Service in Wichita on January 28.

Dr. L. Lafe Bresette, Kansas City, has been named medical division chairman of the United Hospitals Building Fund in that city.

The Arkansas City Medical and Surgical Clinic has announced the dissolution of its partnership, effective February 28. The five physicians in the partnership are Dr. W. G. Weston, Dr. E. R. Maier, Dr. J. V. Sharp, Dr. R. F. Morton, and Dr. James E. Hill.

Dr. S. N. Chaffee, 85, Solomon, who has been in practice more than 50 years, retired from active practice on January 30. A feature story about him and his practice was published by the *Solomon Valley Tribune*.

Dr. Thomas L. Foster, Halstead, directed a program on mental health for the January meeting of the Halstead Parent-Teacher Association.

Dr. LeGrand Byington, Kansas City, has been elected chairman of the health division of the Social Planning Council of Wyandotte County.

COUNTY SOCIETIES

New officers of the Wyandotte County Medical Society, as reported last month, were installed at a dinner meeting held at the Town House Hotel, Kansas City, on January 9. Dr. Glenn R. Peters, retiring president, spoke on "Medicine, 1953," and Dr. J. Warren Manley, new president, discussed "Medicine, 1954." The doctors' wives were guests for the occasion, which was concluded with a dance.

A meeting of the Sedgwick County Society was held at the Lassen Hotel, Wichita, on January 5. Speaker of the evening was Dr. H. A. Schroeder, assistant professor of medicine, Washington University School of Medicine, who discussed "Medical Treatment of Hypertension."

Officers of the Franklin County Society for 1954 were elected at a meeting held at the Ottawa hospital on December 21. Dr. C. W. Henning was elected president; Dr. Louis N. Speer, vice-president, and Dr. James E. Wallen, secretary-treasurer.

A meeting of the Shawnee County Society was held in Topeka, January 4. The speaker was Mr. Doyle Howard, Kansas City, who explained the apparatus, equipment, and methods of administration of oxygen and other gases in therapy.

Members of the Saline County Society were hosts to the Golden Belt Medical Society at a meeting held at the Salina Country Club on January 14. Two Topeka physicians presented the scientific program, Dr. Clyde B. Trees speaking on "Intramedullary Pinning" and Dr. R. M. Brooker discussing "Differential Diagnosis of Surgical Diseases of the Lungs." A dinner and social hour followed the program.

Ground breaking ceremonies were conducted in Wichita last month at the site of the new $90,000 building of the Sedgwick County Medical Society. The ground, at Zimmerly and Hillside, was leased from the Sisters of St. Joseph.

The building, of brick and concrete, will provide auditorium space, executive offices, library, reading room, and quarters for the Medical Service Bureau. It will have approximately 5,400 square feet of floor space on one floor, and there will be off street parking for 200 cars.

Dr. Max W. Wells, Winfield, was elected president of the Cowley County Society at a meeting held at Christmas time at the Winfield Country Club. Dr. Newton C. Smith, Arkansas City, was chosen to serve as vice-president; Dr. R. B. White, secretary, and Dr. W. G. Weston as a new member of the board of censors. A dinner and program, shared with members of the Auxiliary, preceded the business session.

A meeting of the Wyandotte County Society was held at the City-County Health Building, Kansas City, on January 19. Mr. Joe Cohen, an attorney, spoke on "The Doctor in the Courtroom."

A series of health programs is being presented over the air on station KMAN, Manhattan, under the sponsorship of the Riley County Society. The programs have been produced by the American Medical Association.

Dr. J. T. Naramore, Larned, was elected president of the Pawnee County Society at a meeting held on January 14. Dr. T. D. Ewing, Bushton, was named vice-president, and Dr. S. T. Coughlin, Larned, was chosen secretary-treasurer. Dr. LeRoy Shepard, Larned, will be the delegate to the state meeting.

A Blue Cross-Blue Shield membership drive in Coffey County last month was sponsored by the Coffey County Medical Society.

Kansas Licenses to 48

Forty-eight physicians were licensed to practice medicine in Kansas last month, according to Dr. O. W. Davidson, secretary of the Kansas State Board of Medical Registration and Examination. Thirty-nine were licensed by endorsement, and the others successfully completed examinations given by the board in Topeka in December.

Those licensed by reciprocity and the cities in which they indicated they plan to practice are as follows: Berneil Walter Andrews, no location listed; Charles Edward Andrews, Kansas City; Eustace Vernon Ashley, Bushton; James Arnold Bertone, Topeka; John Edmund Birdsell, Topeka; Samuel Beach Chapman, Jr., no location listed; Rudolph Chess, Topeka; Marion O. Christensen, Kiowa; Charles DeHaan, Jr., Pretty Prairie; James Louis Diacon, Wellington; John Lewis Fatland, Council Grove.

Martin James Fitzpatrick, Kansas City; Wayne

Lewis Fowler, Concordia; Evan Lloyd Frederickson, Kansas City; Evalyn Stolaroff Gendel, Topeka; Jack Arnold Haley, Hanover; Edward Albert Heffner, Jr., Hutchinson; John Henry Hege, Lawrence; Lyle Travers Hooker, Osawatomie; Rupert Mitchell Jastram, Hutchinson; Margaret Elizabeth Joyce, Wichita; Lee Franklin Kramer, Cherryvale.

Burritt Samuel Lacy, Jr., Topeka; James Leon McGovern, Wellington; Anthony Wayne Miles, Wichita; John Francis Montague, Topeka; D. Harlan Morgan, no location listed; Van Sam Parmley, Wichita; Edward Bales Price, Jr., Kansas City; Mary Elizabeth Pucci, Manhattan; Norman Keith Pullman, Wichita; William Farthing Sargent, Topeka; Patricia Turk Schloesser, Topeka; Sanford Simon, Kansas City; David Marion Stahl, Kansas City; John Easter Sweeney, Topeka; John William Walker, no location listed; Paul Shan Williamson, Osawatomie, and Karl Arden Youngstrom, Kansas City.

Those licensed by examination are as follows: Harold Altman, Kansas City, Missouri; James Edward Baker, Topeka; Philip G. Danufsky, Kansas City; Joseph Martin Green, Topeka; Lois H. Lohrentz, Kansas City; Richard Lynn Meadows, Topeka; Robert G. Menninger, Topeka; William F. Smith, Jr., Topeka, and Vernon Earl Wilson, Kansas City.

THE KANSAS PRESS LOOKS AT MEDICINE

Editor's Note. In this section the JOURNAL *reproduces editorials relating to medicine which have appeared in the lay press. An effort is made to include both favorable and unfavorable comments, and the Editorial Board in no instance assumes responsibility for the opinions expressed.*

Doctors Don't Run to Washington

A short time ago a *St. Louis Globe-Democrat* reporter interviewed the president of the American Medical Education Foundation, and wrote a strong constructive story about that institution's work. Later, the paper ran an editorial in which it commended American medicine for the prodigious job it is doing in the field of medical education. It also praised the doctors for carrying out the job themselves instead of running to Washington for money.

Many people have been misled into the belief that the medical fraternity is perfectly satisfied with the status quo and is doing little or nothing to improve matters. That idea is 100 per cent wrong. Medical education is an excellent example. Our medical schools are operating under a heavy financial strain. The Foundation was founded in 1950 to do something positive to help. It has raised $2,700,000 from doctors alone. And, along with the National Fund for Medical Education with which it cooperates, contributions totaling $5,100,000 have been obtained during the last three years.

In every phase of medicine, hard work is quietly going on, and it has but one objective—to give the American people better and more extensive medical care. We see this in the endless research to learn more about the cause and cure and prevention of disease. And we see it as well in the search for sound ways and means to lessen the financial shock of illness.

It is true that medicine opposes dictation by government, even as it opposes tax subsidization of activities that are the responsibility of private groups and individuals. That opposition is based on the sure knowledge that the greatest progress for all will come by maintaining medicine in an atmosphere of freedom—not in an atmosphere of political coercion. —*Atwood Citizen-Patriot, January 14, 1954.*

DEATH NOTICES

Edward Jacob Haerle, M.D.

Dr. E. J. Haerle, 65, who had practiced in Minneapolis more than 20 years and who had been Ottawa County health officer for 15 years, died at his home on December 29. A graduate of Southwest School of Medicine and Hospital, Kansas City, in 1912, Dr. Haerle first practiced in Niles. During World War I, he served in the Army medical corps for 14 months. He was an active member of the Saline County Medical Society. Among the survivors is his brother, Dr. Henry H. Haerle, Marysville.

Charles Moses Siever, M.D.

Dr. C. M. Siever, 79, an honorary member of the Shawnee County Society, died at his home in Topeka on January 15. He received his medical degree from Dallas Medical College in 1903 and practiced consecutively in Holton, Texas, and Oklahoma until 1916, when he became head of the health department at Kansas State College. He continued there until 1935, after which he practiced in Holton and Topeka. He was a captain in the medical corps of the Kansas National Guard from 1914 until 1918 and served on the Mexican border and again during World War I.

Clinicopathological Conference

Case Presentation

Dr. Francis N. Lorenz, resident in medicine: This is the first KUMC admission for this patient, a 58-year-old white male, whose chief complaints were a stroke and a lung condition.

He had been in good health except for a chronic, slightly productive cough until five weeks before admission. On July 22, he noted weakness of his left facial muscles, left hand, and left leg, without pain. There was no unconsciousness. He was hospitalized for 10 days and gradually developed some difficulty in talking. Gradual improvement of the weakness on the left side occurred. At dismissal from his local hospital on August 2, the patient could walk with ease but with an obvious left hemiplegic gait. The attention span was said to be poor, but the patient could talk coherently.

Spinal fluid examination done on two occasions at his local hospital showed no blood. X-rays taken at his local hospital showed some pathology in the right lung. It was largely for evaluation of his lung condition that the patient was referred to KUMC.

Approximately two hours before admission, while traveling to KUMC, the patient had a sudden episode in which his eyes became fixed, his color became gray, and there was profuse sweating. He vomited once and then felt somewhat better. Since then, it was his wife's impression that he did not use his right leg as well as previously. Also she noted a peculiar fixed stare in his eyes. He had more difficulty than previously in following instructions. There was no previous history of headache, dizziness, or syncope. There was no history known of hypertension.

The family history revealed one brother to have had moderately severe essential hypertension.

The system review was essentially noncontributory.

On physical examination, temperature was 100° rectally; blood pressure 100/70; pulse 84; respiration 20 and quite irregular. The appearance was that of an elderly male with a fixed stare; he seemed oriented as to time and place but had marked difficulty in following instructions. He would reply to questions with answers sometimes not revealing definite realization of the subject matter.

Head was symmetrical, without masses. The pupils of the eyes were small, regular, and equal, and responded to light and accommodation; the external ocular movements could not be tested because the patient could not follow instructions. It was the impression that the patient could not see. There was bilateral nasal haziness of both disc margins, more marked on the left. The pharynx was clear. The neck was supple.

There were increased breath sounds in the left base of the chest. The left cardiac border of dullness was within the midclavicular line in the 5th interspace. The sounds were of good quality; there were occasional premature contractions. A soft systolic apical murmur was heard but was not considered significant.

The liver, spleen, and kidneys were not palpable in the abdomen. No masses were felt.

There was definite weakness of the left arm and hand. The patient responded fairly well to instruction, but in general he seemed confused and restless. Vision was entirely absent in all fields as far as could be determined. There was left-sided facial weakness with some inability to close the left eye. The patient did not wrinkle the forehead on either side when instructed to do so. His tongue protruded in the midline. Reflexes showed hyperactivity throughout. There was hyperreflexia almost to the point of clonus in the reflexes of his left arm. Hoffman's sign was present bilaterally. No Babinski was present. Abdominal reflexes were absent. There was no ankle clonus.

Laboratory examination of the urine showed it to be acid, sp. gr. 1.019, having a faint trace albumin, no sugar, a few hyalin casts, 2 to 3 pus cells, and 1 to 2 red blood cells per high power field. RBC 4,980,000, hemoglobin 12.8 grams (83 per cent); WBC 11,600, of which 75 per cent were polys, all filamented, 13 per cent lymphocytes, 1 per cent basophil, 11 per cent monocytes. The platelet count was 150,000. On admission, NPN was 27.2, creatine 1.5, sugar 89, serum sodium 105 milliEquivalents per liter, potassium 4.1 mEq., chlorides 95 mEq. One week later NPN was 33, serum sodium 134 mEq., chlorides 98 mEq. On September 15, 1952, the day before death, NPN was 43 milligrams per cent, serum sodium 134 mEq., potassium 5.6, CO_2 21.8 millimols, chlorides 100 mEq. per liter. Cholesterol 388, with 67 per cent esters. Prothrombin time was 75 per cent of normal. Sedimentation rate showed a diagonal curve with a total fall of 3 millimeters in 60 minutes.

While in this hospital, the patient had irregular breathing characteristic at times of both Biot's and Cheyne-Stoke's respiration. The patient was extreme-

From the University of Kansas Medical Center. Edited by Glen R. Shepherd, M.D., and Mahlon Delp, M.D., from recordings of the conference participated in by the departments of medicine, neurology, pathology, radiology, and surgery (neurosurgery), and the third and fourth year classes of medical students.

ly restless, moving back and forth on the bed. The pulse was irregular at times, with bigeminy noted occasionally. The patient was given coramine and small amounts of barbiturate sedation for severe restlessness. Initially the patient took food and fluids fairly well. Marked confusion continued. Blood pressures were 140-150 systolic and 90-100 diastolic. The patient daily had a rectal temperature between 100 and 102°.

Because of his physical condition and confused state, it was impossible to do such further diagnostic studies as a bronchoscopy. Gradually increasing purpuric spots and later ecchymotic areas were noted over the arms, and some were also noted over the face. These were attributed to the patient's constant restless moving about in the bed. Gradually the patient became more restless, requiring greater amounts of intramuscular barbiturates and chloral hydrate. In the last 48 hours, the temperature rose to 103° on several occasions. Respiration became more labored and rapid. There was some dullness to percussion, and decreased breath sounds were present in the right posterior lung field. The day before death, the patient used his right leg much less than previously. On September 16, 1952, the patient expired.

Dr. Delp, chairman: Any questions concerning this history?

Q: Did this patient have a spinal tap while in this hospital?

Dr. Lorenz: No.

Q. Was the patient known to be right- or left-handed?

Dr. Lorenz: Yes, he was right-handed.

Q: Was a serology done?

Dr. Lorenz: No.

Q: What about the terminal episode?

Dr. Lorenz: He was responding enough to take some food and fluids about 9:30 a. m., and about two hours later he had developed cyanosis of the face and left arm with no obvious pain. Then he died.

Q: The history mentions a weakness in the left leg noticed before his admission here. Had that completely cleared up at the time of admission?

Dr. Lorenz: That was hard to evaluate. Probably there was some residual weakness. It was hard to evaluate because of his inability to follow instructions.

Q: Was the patient ever out of bed during the time he was here?

Dr. Lorenz: Yes, initially he was not incontinent. He would by one way or another let his wife know when he had to urinate and she could lead him to the bathroom. But he was out of bed only on those occasions.

Q: In this episode in the car while coming to KUMC, there definitely was no loss of consciousness?

Dr. Lorenz: Not as far as we could tell from his wife's story. There was just the profuse sweating and vomiting without unconsciousness or convulsions.

Dr. Delp: We have one electrocardiographic tracing on this patient. Mr. Culp, do you have that?

Mr. Louis Culp, fourth year student:* The EKG was essentially normal with premature systoles.

Dr. Delp: There are also x-ray studies on this patient. Mr. Claiborne, do you have those?

Mr. Richard Claiborne, fourth year student: PA view shows nothing abnormal in the thoracic cage. The heart is horizontal but seems to be within normal limits. There is nothing abnormal in the great vessels; there seems to be no shift of the mediastinum. The most striking feature is this area of increased density in what appears to be the right upper lobe. There is noted also increased infiltration throughout

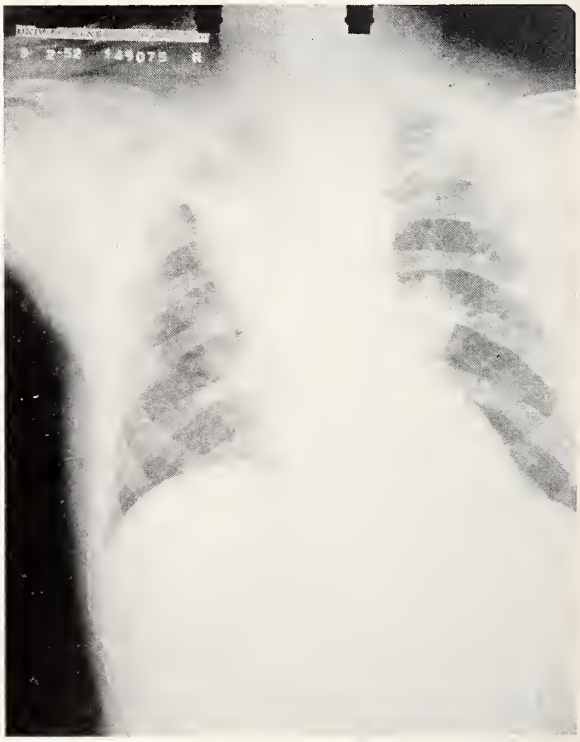


Figure 1. Chest x-ray showing increased density in right upper lobe suggestive of tumor mass or atelectasis, increased markings throughout right lung fields, left hilar shadow increased.

the right lung field as compared to the left. The smooth outline of the area of increased density would suggest the possibility of atelectasis, segmental atelectasis. I cannot rule out a tumor mass, but atelectasis would be my first choice. On the lateral view, you can see an area of increased density. This

* Though a medical student in March, 1953, when this conference occurred, he, like the others referred to as students, received the M.D. degree in June, 1953.

area is sharply demarcated here and would suggest to me the possibility of atelectasis, but I can't rule out tumor mass.

An incidental finding is this compressed vertebra. There is lipping of nearly all the vertebrae, with osteoarthritic changes. I believe these findings are of little importance since they probably produced no symptoms.

Dr. Galen M. Tice, radiology: That is a very good description. The question, of course, is whether this is a tumor mass or atelectasis? I agree with the student that there is a preponderance of evidence in favor of atelectasis. There perhaps is some displacement of the trachea toward that side to go along with it. Peribronchial infection in the right base would be consistent with partial obstruction and thickening of the bronchus and peribronchial infiltration due to retained secretions.

The compression of the vertebra I wouldn't pass off quite so lightly as of no significance. I think it is hard to tell sometimes if a compressed vertebra is of recent origin or if it has been there for a long time. Whether it has been there for weeks, months, or for years may be of some significance in relation to diagnosis.

I can't quite pass by the shadow in the left hilar area. Sometimes the pulmonary artery gives a confusing shadow there; this seems to be a little larger than the average pulmonary artery shadow in a man of this age. There was an increase in bronchial markings above it. That may be a mass in the left hilus. If it is, then that would bring other things into the diagnostic possibilities than we would consider if he had only an area of atelectasis on this side.

Dr. Delp: Mr. Baker, may we have your differential diagnosis?

DIFFERENTIAL DIAGNOSIS

Mr. Floyd Baker, fourth year student: We have here a 58-year-old male who was sent to this hospital primarily for the evaluation of pathology in his right lung found on x-ray studies. However, when the patient arrived at KUMC, his presenting symptoms were those of an intracranial lesion.

First of all, we would like to be able to locate the lesion in his brain, whether it was one focus or several. I want to attempt to describe a location that might explain most of his symptoms.

The original episode, that of weakness of the left side of the face, the left arm, and the left leg, would suggest a lesion in the cortical spinal tract, high up, probably in the cortex or in the internal capsule on the right.

There is some confusion about the second episode because of the difficulty in evaluating the eye signs and because of the entire condition of the patient. It is my impression that this patient probably was blind. This blindness would have to be due to a lesion at or behind the lateral geniculate bodies in the optic radiation, because of the reaction of the pupils to light. The optic radiation runs in the dorsal portion of the internal capsule; going more ventrally and laterally, you get to the arm and leg.

I think it entirely possible that the second episode, which was rather dramatic, could have been due to a hemorrhage with rapid extension across the midline, thereby involving both optic radiations and impinging a little upon the leg area in the internal capsule on the opposite (left) side.

There is just one catch—this man was right-handed and he had some difficulty in talking, and that would indicate that the lesion was in the left temporal region.

In the differential diagnosis one must consider a cerebrovascular accident. The original episode was not that of a hemorrhage. There was no previous history of hypertension. This episode was rather mild, of comparatively slow onset, not dramatic like that usually accompanying a hemorrhage.

An embolus, too, usually is more dramatic. This patient was not fibrillating; about 90 per cent of patients with cerebral emboli are fibrillating. The lung could be a possible source of a cerebral embolus, but that is uncommon.

The original episode was characteristic of cerebral thrombosis, with mild onset and gradual improvement. The second episode was not—it was more like a hemorrhage.

A brain abscess must be considered. We lack a history of any septic course of a previous infectious disease that might have been the source focus of a metastatic brain abscess. However, we still must consider that this might have been an old abscess with the original infection long ago. Usually, with brain abscesses, there are more encephalitis symptoms.

We have to consider tuberculosis because of the lesion in the lung. Usually, intracranial involvement from tuberculosis is more meningitic in character; we had no signs of meningitis in this patient. Also, there would be a more chronic course.

A brain tumor must be considered. A primary brain tumor usually has a more chronic course, with original symptoms of headache and visual disturbances. In the advanced stage there is marked papilledema. This patient did have early papilledema, but it was not marked.

As far as metastatic brain tumor is concerned—there is a very good possibility of a primary tumor in the lung, which is probably the most frequent one to metastasize to the brain. Metastatic brain tumors usually have a more acute onset and a more rapid course than primary brain tumors. This patient was

a male in the right age group for a carcinoma of the lung. He had a chronic cough. Frequently there is metastasis from carcinoma of the lung before there are many symptoms of the primary tumor.

If my localization is wrong, there is always the possibility that the patient had multiple metastases, which would explain the symptoms.

We always must consider the possibility of two diseases present in the same patient. For example, tuberculosis of the lung and cerebrovascular accident. But, whenever there is good evidence that the patient has one disease and all the symptoms may be explained on the basis of the one disease, I incline to that diagnosis until proved otherwise.

Therefore, my diagnosis is bronchogenic carcinoma with metastases to the brain. That vertebra doesn't appear to have had very much destruction, but there is the possibility of extension to the vertebra.

As for the cause of death—I feel that this patient died of pneumonia with probable respiratory failure. I think it should be mentioned that this patient was in respiratory difficulty all the time he was here. He was receiving heavy doses of barbiturates that might have had some effect on his respiration.

Dr. Delp: Mr. Blim, how do you account for these neurological findings?

Mr. Richard D. Blim, fourth year student: I think they are easiest to account for on the basis of diffuse multiple metastases. If I had to pinpoint them, I would pick the internal capsule, but that doesn't explain the aphasia or confusion the patient had. Some of the confusion could have been due to the chloral hydrate or the barbiturates he was receiving.

Dr. Delp: Mr. Black, how do you explain this patient's fever? He had fever at the time he arrived; he had fever each day he was here.

Mr. Don Black, fourth year student: I can't explain why he had fever when he arrived. He had fever while here probably because of aspiration of fluids.

Dr. Delp: Mr. Battin, do you have any suggestions?

Mr. Keith Battin, fourth year student: I think probably the fever was due to a pneumonic process.

Dr. Delp: Mr. Altman, do you think the patient had a metastatic disease of the brain?

Mr. Harold Altman, fourth year student: I think you probably could explain part or all of his terminal fever on the fact that he was extremely restless and was moving around so much.

Dr. Delp: Do you think that he had metastatic disease of the brain?

Mr. Altman: Yes.

CLINICAL DISCUSSION

Dr. Delp: We are going to ask Dr. Steegmann and Dr. Brackett to localize the central nervous system lesion for us. You might comment on the etiology, too, if you care to. Dr. Steegmann?

Dr. A. T. Steegmann, neurology: I do not find the localization easy in this case. We are more or less forced to assume that probably there was a lesion on both sides. I feel that the symptoms point more to a lesion in the right frontal region, involving the motor portion of the hemisphere, because there was left-sided weakness. The patient had also what I feel was hemianopsia, but it is difficult to delineate that because of the difficulty in examining the patient.

After thinking it over and after seeing the patient, I decided that this so-called blindness probably was an apperceptive blindness—what sometimes is spoken of as Pick's blindness—which really is a form of disorientation. It is not actually due to an involvement of the visual pathways but is an involvement of the associational visual mechanisms. These patients appear to be blind, they are not actually blind. That, of course, would carry with it the thought that there might have been a lesion in the parietal lobe, perhaps on either side, but probably in the dominant hemisphere. The patient was right-handed, if I am not mistaken.

Then the appearance of symptoms referable to the right side brought up the question of whether there was a lesion on the opposite side, also. It was my opinion, in view of the changes in the lung, that this probably was metastatic, but there was one difficulty. That was the relatively sudden onset of symptoms. So I changed my mind as to possible metastasis and ended up my consultation note, on the first consultation, by being a bit more conservative and saying that it might be a vascular lesion.

I still think the question of precise localization in this case is difficult. I would like to make one comment about the question of localization. There is a difference between localization of a hemiplegia secondary to an occlusion of the sylvian artery and a hemiplegia from the internal capsule. I notice that students frequently speak of a hemiplegia from the internal capsule in the same sense that they speak of a hemiplegia from an occlusion of the branches of the sylvian artery distal to the perforating branches. The hemiplegia due to a lesion in the perforating branches is always symmetrical, being equally in the arm and leg, is spastic in type, and is not associated with any aphasia or aphasic disturbances.

On the other hand, if the lesion is in the distal portions, encroaching upon the sylvian artery distal to the perforating branches, then the hemiplegia is of arm-and-aphasia type. It is much more pronounced in the arm than in the leg.

If it is a hemiplegia involving the perforating

branches, encroaching somewhat on the island of Reil but not affecting the superficial branches in the cortex, then the hemiplegia is symmetrical but usually flaccid. Those are important differential diagnostic ideas that I think everyone should know. It only means that you must know a little about the blood supply in the brain to understand it.

Dr. Delp: Dr. Brackett?

Dr. Charles Brackett, neurosurgery: I think Mr. Baker regarded the first episode just right and that the patient had an involvement of the cortical spinal tract, the cortical bulbar tract, somewhere above the 7th nerve nucleus. That's about all you can say as to localization of the first lesion.

In the hospital he was thought to be blind. This would imply one of two things: he had bilateral occipital lobe lesions or lesions of the radiations. That could not be a simple lesion; it was bound to lie in two separate structures. Or he had a lesion in the left parietal-occipital area (being plainly right-handed) so that he had a visual agnosia—did not recognize what he saw.

But to be perfectly correct, I believe it was in the lateral geniculus because he had a light reaction from it.

And, finally, when he died, he did have weakness of the right leg. So I note that the patient had evidence of three localizations: right frontal, left parietal-occipital, and possibly the brain stem.

As to the nature of the process—this patient awoke in the morning with a left facial paralysis which gradually progressed over a period of hours to involve the arm and the leg. He had no hypertension, he had no headache, no unconsciousness, no evidence of increased pressure; and the spinal fluid was normal on several occasions. So I think this was clearly an episode of thrombosis.

The second episode, on the way in to KUMC, probably was not a thrombotic episode.

The main problem in the case is the differential diagnosis between metastatic carcinoma from the lung and cerebral thrombosis. There is nothing in the history to suggest embolization.

Metastases from the lung are embolized. They may produce a sudden onset occasionally, due either to an embolization or to hemorrhage from the tumor. But I think it would be extremely unlikely for three such episodes to occur. Furthermore, following each episode there was significant improvement, so our conclusion was that this patient was suffering from cerebral thromboses unrelated to his chest pathology.

Dr. Delp: Dr. Schloerb, with the history, ignoring the neurological findings and the other physical findings, plus this x-ray film, do you believe the patient had a carcinoma of the lung?

Dr. Paul Schloerb, surgery: Yes. One question that comes to my mind: He did develop purpura during the course of his hospital stay, and I wonder if that was evidence in favor of there being hemorrhage into the cerebral metastasis? Was that purpura studied in terms of bleeding time and clotting time?

Dr. Lorenz: The patient did have a normal prothrombin time. No other studies were made.

Dr. Delp: Dr. Berry, assuming that this is a carcinoma of the lung, did this patient die of the carcinoma of the lung?

Dr. Max Berry, medicine: No, I don't think so. He had fever all the time. That, of course, is sometimes a complication of the cerebral lesions, but it should have been a little higher than that.

Dr. Delp: Do you believe this patient had carcinoma of the lung with metastasis?

Dr. Berry: To the brain? It is awfully difficult to explain it otherwise, because of three things. First, he was well until he started out with these brain episodes, except for a chronic cough which nobody made much fuss about. The second thing is that he had a lesion in his lung. We can see it on the x-ray film. It probably was atelectasis, with bronchial obstruction due to the tumor. The third thing is, he had three different localizations in his brain which would mean that something could be seeding from the lung.

Now, carcinoma is one of the things it could have been. And the other thing that it could have been was an abscess or infection in his lung.

But he had a lesion in the back with a crumpled vertebra, and with all those things tying together, you are forced to make a diagnosis of carcinoma of the lung.

Dr. Delp: Dr. Allen, why did this patient have fever?

Dr. Max Allen, medicine: We thought his fever was due to the lesion in his lung—atelectasis and probably bronchial pneumonia and perhaps abscess formation back of the obstruction in the lungs.

Dr. Delp: Is there anyone in the audience who would like to volunteer a statement as to why this patient had fever, and what the lesion was in the brain? Dr. Rankin?

Dr. Thomas J. Rankin, medicine: There is one thing I would like to know. What was the pulse rate?

Dr. Lorenz: It was not phenomenal; it was 84 on admission.

Dr. Rankin: I must go along with the majority and say that I thought this patient had carcinoma of the lung with metastasis to the brain. However, I feel that the possibility of a metastatic abscess to the brain from pulmonary infection has not been given enough consideration.

Before the present frequent use of antibiotics, I

have seen such cerebral abscess formation from pulmonary disease fairly frequently. Since the antibiotic era, this has not been my experience.

Dr. Delp: How many people think this man had a carcinoma of the lung? How many think he had metastatic carcinoma of the brain? Does anyone think he had any other sort of metastatic disease of the brain?

Dr. Rankin is the only one who suggested that he had an infectious metastatic lesion, I believe.

Dr. Charles C. Herbert, medicine: The possibility of multiple emboli to the brain is suggested by the clinical picture. The question is the source. The description of the heart suggests a normal heart, so we have no evidence from what we have been given to indicate that he may have had an endocarditis. It is usually a hidden disease in old men, or it can be. The fact is that there has been so much talk about the cause of the fever that I am beginning to suspect the rare.

Dr. Delp: Dr. Frenkel, may we have the pathology report?

PATHOLOGY REPORT

Dr. Jacob K. Frenkel, pathology and oncology: Autopsy demonstrated the occurrence of two lesions, both rather rare. As was suspected, this patient had a carcinoma of the lung. However, the carcinoma was not well seen grossly and looked very much like pneumonia. It extended through both lobes, and there was considerable discussion at the time of autopsy as to whether this was carcinoma or whether it was pneumonia.

We will see from its microscopic appearance that the lesion was a relatively unusual one, and that it somewhat resembled both alveolar cell carcinoma and pulmonary adenomatosis in that the distribution of the carcinoma followed the alveolar lining cells.

The second lesion that was found grossly was a lesion on the mitral valve, a form of endocarditis not associated with bacteria, so-called nonbacterial verrucous endocardiosis. This lesion, to my knowledge, does not commonly give rise to emboli. In a survey of some 50 to 75 cases in the literature, no emboli are mentioned. However, Dr. Pollack informs me that she has seen one or two cases where emboli were present. Emboli from the mitral valve, nonseptic emboli, give rise to infarcts in the brain, heart, and kidney.

The lesions in the brain had their distribution in both the left temporal and the right occipital lobes, and in both the posterior and the central gyri of the brain. The patient also had bronchopneumonia.

Microscopic: As we see on this slide, the patient had a carcinoma with papillary as well as squamous components. The predominant form is the papillary type. This resembles somewhat an infectious disease of sheep, jaagsiekte, thought to be due to a virus. This disease is endemic in sheep and, in at least one instance, pulmonary adenomatosis has been found in a woman who had spent some time on a sheep ranch where that disease was prevalent. No such association is recorded in the history of our patient; his occupation was that of brickmason.

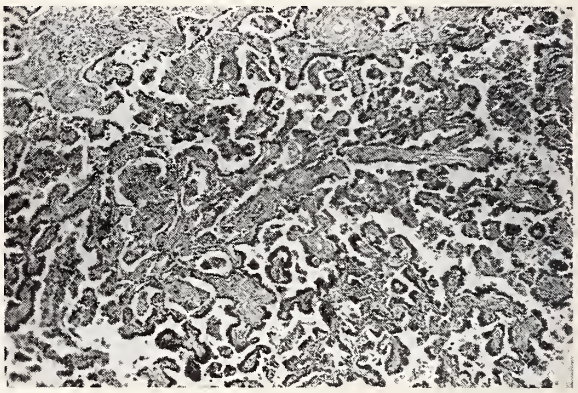


Figure 2. Lung with papillary carcinoma predominating; some squamous areas are also present. Magnification x(92).

This tumor might be classified also as an adenocarcinoma of the lung, because the tumor cells resemble the cells of the small bronchioles. No mucus was found. Pulmonary adenomatosis is often multicentric in origin, and papillary bronchogenic carcinoma can be likewise. The presence of diffuse involvement of the lung may also be explained by the occurrence of metastasis throughout the lung.

This next slide shows a case of pulmonary adenomatosis from the woman referred to. The following slide shows a lesion found in the lungs of a sheep with which this woman had been associated.



Figure 3. Mitral valve with vegetation, composed of fibrin and platelets. Note the absence of bacteria and leukocytes. Magnification x(25).

Here we see the lesion of the mitral valve; here is the valve leaflet. The entire lesion has been sec-

tioned so it is missing from the gross specimen. Here is the thrombotic projection, the verruca, on the valve. You will notice its uniform appearance and the absence of bacterial colonies such as are found frequently with bacterial endocarditis.

Here we see a lesion in a cerebral vessel showing embolic material, and a focus of softening in the adjacent brain. In the heart, the embolus of the non-bacterial type results in an infarct.

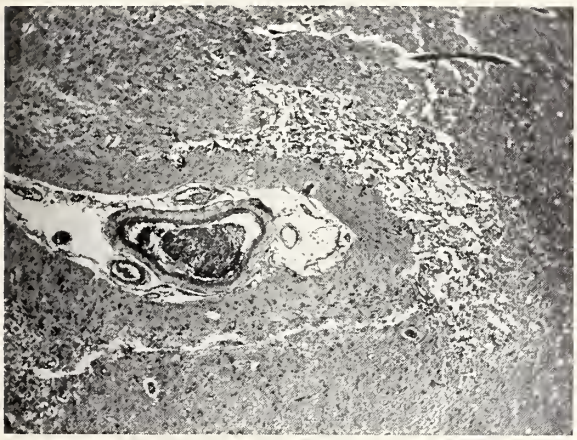


Figure 4. Brain showing organizing embolus in vessel and adjacent area of infarction. Magnification x(55).

In summary, we saw a concurrence here of two fairly rare lesions, producing a rather bizarre picture in this patient who puzzled us very much.

Dr. Delp: Any questions of Dr. Frenkel?

Dr. Tice: Was there pathology in the left lung?

Dr. Frenkel: Yes, the tumor was in both lungs, and it was alveolar in distribution; bronchopneumonia likewise was present.

Dr. Berry: Did you examine that dorsal vertebra that was compressed?

Dr. Frenkel: It was examined. It seemed to be the result of an old fracture. It did not contain metastatic tumor.

Dr. Robert Bolinger, medicine: Dr. Frenkel said that there was embolus in the coronary artery. How did he know whether it was embolus or thrombosis?

Dr. Frenkel: The appearance of the thrombus suggested that it arose as an embolus.

Q: Were there lesions in the hypothalamus?

Dr. Frenkel: No.

ANATOMICAL DIAGNOSIS

Primary: Papillary bronchogenic carcinoma of the upper lobe of the right lung with metastases to the lower and middle lobes of the right lung and both sides of the left lung.

Bronchopneumonia of the lower lobe of the right lung and both lobes of the left lung.

Non-bacterial verrucous endocardiosis of the mitral valve with embolic occlusion of small branches of the coronary arteries and multiple small infarcts of the myocardium.

Multiple infarcts of the left kidney, of the left temporal and right occipital lobes, and of both posterior central gyri of the brain.

Focal fatty metamorphosis of the liver.

Accessory: Arteriosclerosis of the right circumflex and left anterior descending coronary arteries, advanced; of the left circumflex coronary artery, slight; of the cerebral arteries, slight to moderate; of the aorta and the pulmonary arteries, slight.

Sclerosis of the bases of the cusps of the mitral and aortic valves.

Fibrous thickening of the appositional surfaces of the mitral valve and of the tricuspid valve, slight.

Chiari's network, right atrium.

Hypertrophy and dilatation of the heart (400 grams—formalin-fixed).

Focal fibrosis of the myocardium of both ventricles.

Chronic passive congestion of the liver and of the small and large bowel.

Multiple ecchymoses of the mucosa of the small and large intestines.

Hydropericardium (50 ml.).

Fibrosis of the spleen.

Scar of lower pole of right kidney.

Cysts of right kidney, small.

Absence of the teeth.

BIBLIOGRAPHY

Liebow, A. A.: Tumors of the lower respiratory tract, Atlas of Tumor Pathology, Section V, Fascicle 17, pp. 189-249 ill., 1952.

Allen, A. C., and Sirota, J. H.: The morphogenesis and significance of degenerative verrucal endocardiosis (terminal endocarditis, endocarditis simplex, non-bacterial thrombotic endocarditis), Am. J. Path. 20:1025-1055 (Nov.) 1944.

Hildebrand, E.: Pulmonary adenomatosis, report of a case, Am. Rev. Tuberc. 57:281-286 (Mar.) 1948.

There seems to be little doubt that physical and intellectual fatigue, as well as painful emotions, decrease resistance of the body to infection; but the physiologic mechanisms which determine these important relationships are totally unknown. Despite all this ignorance, however, a belief shines as a beacon to guide our efforts. It is that the defense mechanisms of the body which promote resistance to infection appear to be closely related to the physiologic processes which make for good health—good health being here measured in the terms broadly referred to as well-being by the layman. The inner logic of the program of antituberculosis associations should lead them to become truly health associations. For it can hardly be doubted that, in final analysis, health is a positive attribute rather than merely the absence of disease.—*Rene J. Dubos, Ph.D., Am. Rev. Tuberc., July, 1953.*

The Cerebral Circulation: Comments on Cerebral Angiospasm

John O. Mansfield, M.D.

Honolulu, Hawaii

I. INTRODUCTION

The cerebral circulation is certainly not a new phase of physiology, but one that has been the subject of investigation for more than a century.[22] The literature on this subject is quite voluminous, especially in the past 25 years. World War II brought about a great increase in man's knowledge of cerebral circulation due to its importance and application to the field of military activity, particularly that of high-speed, high-altitude aviation. The year 1944 marked a symposium on the cerebral circulation,[1, 4, 10, 22] an evaluation of the then current status of cerebral circulation.

Here, Schmidt noted, "The most important human strains of combat flying are related to anoxia, aeroembolism, cold, acceleration, motion sickness, disturbances of the special sensory apparatus, fatigue, and psychic deterioration. This list is not likely to require substantial modification when more recent experience is disclosed. Now it is a striking fact that the brain is the limiting factor with respect to all these strains, either because its functional capacity is reduced (as by anoxia, cold, or the cerebral anemia or congestion associated with acceleration), or because it is subjected to an intolerable bombardment by sensory nerve impulses (as in the pain of decompression sickness, the nausea of motion sickness, the discomfort of cold, or the accumulated psychic trauma associated with combat aviation). The blood supply of the brain then becomes a matter of immediate concern, not only because here, as elsewhere in the body, the capacity of the tissue to function is limited by the capacity of its circulation to meet its changing requirements, but for the additional special reason that the brain is unable to contract much of an oxygen debt and therefore is more subject than other tissues to derangement by an insufficient supply of blood."

Recent advances in neurosurgery have also stimulated the need for more knowledge of the cerebral circulation.

Since the symposium in 1944, new experimental techniques have greatly added to our knowledge of the circulation of the brain. The nitrous oxide method of Kety and Schmidt has opened a whole new phase of investigation, produced much additional information, and promises more in the near future.[2, 9, 13, 14, 15, 16, 17, 23, 25, 26]

It is the purpose of this paper to discuss briefly the cerebral circulation, with some comments on cerebral angiospasm.

II. ANATOMY—GROSS

The general arrangement of the cerebral vessels is quite constant in different mammals although the details show wide species variations.

Arterial:[23] The arterial supply of the brain is derived from the carotid and vertebral arteries, the main branches of which fuse at the base of the brain to form the circle of Willis. From this arise three pairs of arteries (anterior, middle, and post cerebral) which course over the convex surfaces of the cortex above the tentorium and send smaller branches into the brain substance constituting its main supply of blood. The basal ganglia and choroid plexus are supplied by a few direct branches. The anterior spinal artery forms an anastomosis with the vertebral-basilar system which is functionally insignificant in man but may be of considerable importance in some animals (particularly dogs).

The size of the primary arteries to the brain, and hence the importance, may vary considerably in different species. Thus the carotids are practically the exclusive supply of the brain of the ox and goat. In the lemur, the carotids are rudimentary and the vertebrals are the main source of blood supply. Man is intermediate in this respect, and both carotids and vertebrals are important in supplying blood to the brain. The monkey is similar to man in this respect. The rabbit is the only common laboratory animal in which the main supply of the circle of Willis is the internal carotid. The main supply in the dog and cat is a complex anastomosis between the orbital distribution of the external carotid and the internal carotid just before it enters the circle of Willis. Here, the internal carotid is relatively insignificant until just before it enters the circle inside the skull.

The significance of these anatomical arterial variations is far-reaching in experimental attempts to measure cerebral arterial flow by means of instru-

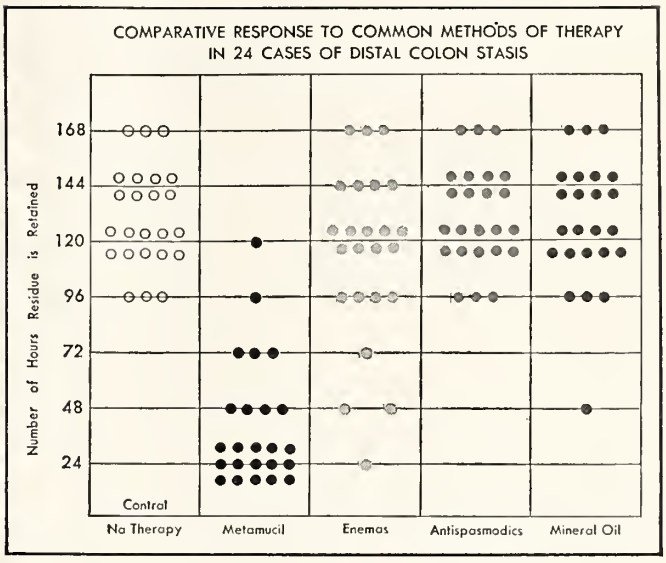
---

This is one of 11 theses, written by fourth year students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group judged to be the best by the faculty at the school.

Thesis written while the author was a fourth year student. Dr. Mansfield is now interning at U. S. Army Tripler General Hospital, Honolulu, Hawaii.



Distal Colon Stasis



Management of Distal Colon Stasis with Metamucil®

The "irritable colon" resulting in distal colon stasis is a hard-to-manage by-product of many abdominal or stress conditions.

Roentgen evaluation of the commonly used methods to combat colonic stasis has shown the value of Metamucil because of its lack of irritation and its high degree of effectiveness* in this most prevalent type of stasis.

Metamucil is the highly refined mucilloid of Plantago ovata (50%), a seed of the psyllium group, combined with dextrose (50%) as a dispersing agent. It produces smooth fecal bulk necessary to incite the normal peristaltic reflexes, without causing irritation, straining, impaction or interference with the digestion or absorption of vitamins.

The average adult dose is one teaspoonful of Metamucil powder in a glass of cool water, milk or juice, followed by an additional glass of fluid if indicated. This amount of fluid is essential for the production of "smoothage."

It is supplied in containers of 4, 8 and 16 ounces. Metamucil is accepted by the Council on Pharmacy and Chemistry of the American Medical Association.

SEARLE *Research in the Service of Medicine*

*Barowsky, H.: A Roentgenographic Evaluation of the Common Measures Employed in the Treatment of Colonic Stasis. Rev. Gastroenterol. *19*:154 (Feb.) 1952.

ments applied to the carotid and/or vertebral system. In this procedure it is necessary that all communications between the intra- and extracerebral distributions of these vessels be closed, otherwise there is no way of determining whether changes in flow are due to the intracerebral or the extracerebral systems.[23]

The brain in man is supplied by blood entirely by means of the internal carotids and vertebrals. There is no significant anastomosis with extracerebral arteries as exist in lower mammals. The carotids supply (in general) the anterior and middle portions of brain on each side. The basilar artery (formed by the vertebrals) supplies the occipital lobes, portions of the temporal lobes, and the structures of the posterior fossa.[13]

By means of dye injection techniques in living human subjects, there is good evidence that blood entering the brain via each internal carotid artery is distributed almost wholly to the ipsilateral hemisphere and drained predominantly by the corresponding internal jugular vein.[24] There is also good evidence that blood in each jugular is fairly representative of the drainage from all histological components of the brain. Blood obtained from the superior bulb of the internal jugular vein is relatively free of blood derived from extracerebral sources.[24] However, surgical procedures involving the ligation of the carotid[26] have demonstrated the potential contralateral arterial supply in human beings.

Venous:[23] The venous return from the brain is accomplished through vessels of three groups—superficial cerebral veins, deep cerebral veins, and cerebral venous sinuses. The superficial cerebral veins empty mainly into the superior longitudinal and lateral sinuses; several larger channels empty into the superior sagittal sinus. The deep cerebral veins are gathered into the paired internal cerebral veins which unite to form the great cerebral vein (Galen) which drains into the straight sinus. The cerebral venous sinuses are clefts in the dura mater and are devoid of elastic and muscle tissue ordinarily seen in veins; the most important of these are the unpaired superior and inferior sagittal sinuses, the paired lateral sinuses, the unpaired straight sinus, the paired cavernous sinuses, the occipital sinus. Five of these (the superior sagittal, straight, occipital, and the two lateral sinuses) usually unite at a point directly under the external occipital protuberance to form the torcular Herophili.

Venous blood leaves the brain mainly by the jugulars, into which the torcular and cavernous sinuses drain directly. There are also communications with the veins of the spinal canal, which are arranged as anterior and posterior plexuses communicating at each intervertebral space with the vertebral, intercostal, and lumbar venous systems. The small emissary veins communicate directly between the cerebral sinuses and the extracerebral venous system of the head.

The major venous return from the brains of primates and man is through the internal jugulars, although the external jugulars also carry appreciable amounts. The converse is not true: the blood in the superior jugular bulb is contaminated only with negligible amounts (average 3 per cent) of blood derived from extracerebral tissue. It is important to note, however, that once the large common facial vein enters the internal jugular this is no longer the case.[23]

The Munro-Kellie Doctrine:[23] The Scotch anatomist, Munro, formulated the conception that since the cranium is a rigid, indistensible box completely filled with incompressible contents, the quantity of blood contained within it cannot change appreciably. Kellie supported this hypothesis 40 years later by experiments and autopsies.

The significance of this hypothesis is that it has several important consequences. Increase in the volume of any one of the contents of the cranium will be at the expense of the others. The most labile of the contents being cerebrospinal fluid (CSF) and blood, one or both of these will be displaced by increase of normal or abnormal tissue in the skull. Also an increase of the CSF pressure is transmitted equally throughout the system, and, once it surpasses the pressure in the cerebral blood vessels, they will collapse until their pressure is at least equal to that exerted on their walls. Since the intravascular pressure is lowest in the veins, as long as there is an outward flow of blood the venous pressure must equal that of the CSF, and an increase in CSF pressure will be accompanied by an increase in venous pressure. A venous pressure rise will also result in a rise in CSF pressure.[23]

Anatomical problems:[1] Organs with pedicles through which the major blood vessels enter and leave are the most favorable for the study of blood flow. This is not so with the brain, for here the blood supply is quite complex and there are a number of anastomoses.

If the rate of flow is to be measured on the arterial side, an experimental animal is required in which it is possible to include all the blood going into the brain and exclude all going elsewhere.

That the vertebral vein plexus is of considerable importance is shown by Batson[1] in a case of total occlusion of the superior vena cava where the patient's only complaint was a swollen face. Here it must be admitted that the plexus must have been a functioning system in order to be able to take over in the face of this emergency.

Some animals have a peculiar elaboration of vessels known as a rete mirabile. This network provides

Sixth Annual Mid-West Cancer Conference

Broadview Hotel, Wichita, Kansas April 1 and 2, 1954

GUEST SPEAKERS

A. N. Arneson, M.D.
St. Louis, Missouri

Louis H. Clerf, M.D.
Philadelphia, Pennsylvania

Warren H. Cole, M.D.
Chicago, Illinois

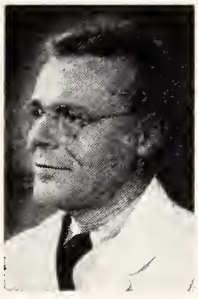
Hugh F. Hare, M.D.
Los Angeles, California

N. L. Higinbotham, M.D.
New York, New York

Richard H. Overhold, M.D.
Brookline, Massachusetts

Cornelius P. Rhoads, M.D.
New York, New York

Arthur P. Stout, M.D.
New York, New York



Cornelius P. Rhoads, M.D.

Director, Memorial Hospital, New York City; graduate, Harvard Medical School, 1924; Fellow, American College of Physicians, American Association of Pathologists and Bacteriologists, American Society for Experimental Pathology, American Radium Society.



Louis H. Clerf, M.D.

Professor of Laryngology and Broncho - esophagology, Jefferson Medical College, Philadelphia; Graduate, Jefferson Medical College; Diplomate, American Board of Otolaryngology; Fellow, American College of Surgeons, American College of Chest Physicians.

Biographical sketches and pictures of additional speakers will be featured in subsequent issues of the Journal.

Sponsored by
American Cancer Society, Kansas Division
Committee on Control of Cancer, Kansas Medical Society

intra- and extra-cranial junctions between the cerebral vessels and the external carotids. These animals are unsuited for studies of cerebral arterial flow. The Rhesus monkey does not have a rete mirabile. Here, for practical purposes, the anatomy approximates that of man. The cat and dog are not suitable because of retes.

Of the animals reviewed (cat, dog, monkey, rabbit), the venous side is unsuited in all for the study of the cerebral blood flow.

Histological anatomy: The morphology of the cerebral blood vessels differs from that of the blood vessels of the visceral organs. The intima is separated from the media by a distinct, elastic membrane which is better developed here than in the blood vessels elsewhere in the body.

The media likewise contains a few elastic fibers irregularly scattered and intimately mixed with the circularly-arranged muscle fibers. No external elastic membrane between media and adventitia is present in cerebral blood vessels. The adventitia consists of loose connective tissue, the nuclei of which are parallel to the axis of the blood vessel. There is a lack of elastic tissue in the poorly developed adventitia.[21]

The elastic tissue of the cerebral vessels is thus segregated into a well developed layer which is relatively rigid, and it is surrounded by a smooth muscle layer, probably devoid of elastic tissue. This muscle layer, in turn, is surrounded by a sponge-like perivascular space which, too, is non-rigid. This arrangement is in contrast to the blood vessels in skeletal muscle, for example, where the elastic tissue is interspersed throughout the blood vessel walls, thus affording comparatively slight shock absorbing quality.[21]

The Virchow-Robin perivascular space, a typical feature of the cerebral vascular system, is a structure of pathologic importance.[21] It may be looked upon as a loosened adventitia lined with flattened connective tissue cells. This space becomes visible under pathologic conditions, for normally it is collapsed and shrunken. The Virchow-Robin space is peculiar to the central nervous system, apparently taking the place of a lymphatic system, for there are no separate lymphatic vessels in the brain or spinal cord.

The space of His outside the adventitia is considered generally as an artifact. The space of Held is an area of tissue rarefaction in the parenchyma of the brain.

In regard to perivascular nerves, Humphreys[12] in 1929 demonstrated by histologic techniques a rich perivascular nerve supply in nearly 100 per cent of the intra-cerebral vessels impregnated. These vessels were of all calibers down to 10 microns. Fiber counts gave an estimate of this rich supply. He believed these nerves to be autonomic and vasomotor—both sympathetic and parasympathetic. This is discussed in more detail later.

III. PHYSIOLOGY

There is now a mass of unequivocal evidence from experiments on animals to indicate that the blood vessels in their brains are subject to all the influences that affect the circulation elsewhere in the body, viz., vasoconstrictor and vasodilator nerve impulses, metabolic products, hormones, and drugs. A smaller amount of evidence indicating that some of these relations also exist in man has become available very recently and more is to be expected in the near future.[23]

Blood pressure: In general the cerebral circulation tends to follow passively the changes in arterial pressure, even to a greater extent than most other organs; however, it probably has a greater capacity for resisting or compensating such changes than has been suspected in the past.[23]

According to Kety,[13] the rate of cerebral blood flow depends on two factors: (1) blood pressure head, and (2) cerebrovascular resistance, each of which may be influenced by a variety of factors. His work does not substantiate the earlier belief that cerebral blood flow passively follows changes in arterial blood pressure. "The cerebral blood flow is, above a critical minimal arterial pressure, regulated intrinsically. The critical minimal arterial pressure is probably not far below the normal mean pressure, as indicated by the cerebral symptoms associated with orthostatic hypotension. Above shock levels of blood pressure the cerebrovascular resistance is the predominant regulator of the cerebral blood flow. Cerebrovascular resistance is the resultant of all factors tending to impede the flow of blood through the brain and includes intracranial pressure, blood viscosity, and organic changes as well as the functional tone of cerebral vessels."

Vasoconstrictor nerves: Forbes and Wolff[7] furnished the first unequivocal demonstration of constriction of cerebral blood vessels resulting from the stimulation of sympathetic nerves. This was done by microscopic observations and measurements of the surface vessels of the parietal cortex of anesthetized cats. These decreases in vessel diameter were in the order of 10 per cent or less, whereas corresponding decreases in dural vessels were about 30 per cent and those of the skin 80 per cent. The cerebral effects were obtained by stimulation of the ipsilateral cervical sympathetic nerve.

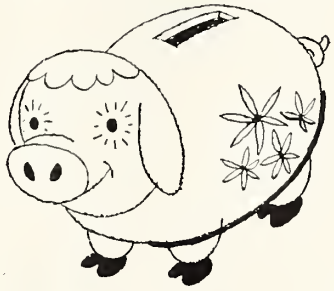
No effects were obtained by stimulation of fibers of the stellate ganglia and the vessels of the vertebral arteries. Cervical sympathetic cerebral vasoconstrictor innervation has been demonstrated also, by C. F. Schmidt et al. in a series of papers, by means of

FOR ALL INFANT FEEDING

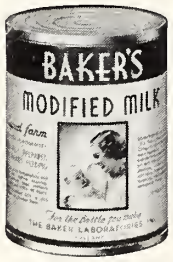
Baker's Modified Milk

FILLS THE NEED FOR...

Economy

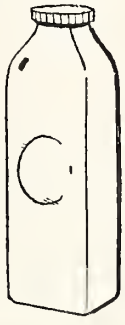


Simplicity



+ H_2O

Quality



MADE FROM GRADE A MILK

Baker's Modified Milk is made from Grade A Milk (U. S. Public Health Service Milk Code), which has been modified by replacement of the milk fat with animal and vegetable oils and by the addition of carbohydrates, vitamins and iron.

BAKER'S MODIFIED MILK

THE BAKER LABORATORIES INC.

Milk Products Exclusively for the Medical Profession

Main Office: Cleveland 3, Ohio
Plant: East Troy, Wiscansin

Division Offices: Atlanta, Dallas, Denver, Greensboro, N. C., Los Angeles, San Francisca, Seattle

ACCEPTED COUNCIL ON FOODS AND NUTRITION AMERICAN MEDICAL ASSOCIATION

a thermo-couple inserted in the hypothalamic region and parietal cortex of the cat and the parietal cortex of the rabbit. However, the same method revealed no sign of innervation in the medulla, pons, and occipital cortex of the cat. This made evident that generalizations of the cerebral circulation as a whole could not be drawn from observations on only one part of it, and that it would be necessary to obtain data of the behavior of the cerebral circulation as a whole. This was done by direct measurements of the cerebral arterial inflow in monkeys, and no significant reduction in cerebral flow was obtained by sympathetic stimulation strong enough to produce mydriasis and salivation.

The possibility of cerebral angiospasm from sympathetic impulses or from intrinsic vasoconstrictor innervation with or without extracranial connections can neither be affirmed nor denied, according to Schmidt,[23] but at present the evidence is against it.

Vasodilator nerves: One of the most important peculiarities of the cerebral circulation is that its vessels can be dilated strongly by many influences, constricted weakly by only a few; the best available conception of its intrinsic control is that it is dilator rather than constrictor in direction.

The first definite evidence of cerebral vasodilator innervation was demonstrated by means of the pial window, as was vasoconstrictor innervation, and this is mediated by nerve fibers passing from the facial nerve through the geniculate ganglion by way of the superficial petrosal nerve to the pericarotid plexus.[3] Other pathways such as the vagodepressor nerve and the carotid sinus nerves have been claimed to be cerebral vasodilators; however, the evidence for this is apparently not well founded.[23] The significance functionally of the first mentioned paths is not yet known.

Metabolic products: In general, as with the other vessels in the body, the cerebral vessels are dilated by all the usual products or consequences of metabolism. This includes decreased oxygen, increased carbon dioxide, increased acidity, increased temperature, as well as a number of other agents such as acetylcholine, histamine, potassium, and calcium ions which are now associated with cellular activity.[4, 13, 23, 25] There is also cerebral vascular constriction produced by reverse metabolic changes such as increased oxygen and decreased CO_2. Decreased CO_2 is probably the most potent cerebral vasoconstrictor yet found and used under physiological conditions. In this respect the cerebral vessels are unique. Schmidt[23] believes that the degree of dilatation in severe anoxemia in man would be nearly equal to that of increased CO_2, although the effects of severe anoxemia (i.e., less than 10 per cent O_2) have not yet been studied. Schmidt also believes that the behavior of the cerebral circulation under physiological as well as pathological conditions is influenced more by metabolic agents than by any other factor.

It has been known for some time that acids dilate the cerebral vessels and alkalis produce constriction. These changes, however, are not as marked nor as consistent as changes produced by variation in CO_2 tension. The most pertinent evidence has been obtained recently in studies on patients in diabetic acidosis. Here the cerebral blood flow apparently decreases with arterial CO_2 tension over the arterial pH range 7.40 to 7.10. When the acidosis becomes more severe than pH 7.10, cerebral blood flow tends to increase sharply, the curve of its behavior being very similar to that portraying pulmonary ventilation.[17] This is consistent with the belief that CO_2 tension is the dominant influence in regulating both the tone of cerebral vessels and the activity of the respiratory center over the ordinary physiological range. Beyond this, however, other agents may overrule the normal regulator.

Hormones: Wiggers, as far back as 1905, demonstrated in the perfused isolated dog's brain the vasoconstriction response and diminution in the perfusion flow to epinephrine, when it was added to the perfusion fluid. That epinephrine can constrict the cerebral blood vessels has been confirmed by many other investigators using various techniques on dogs, cats, and monkeys. However, there is general agreement that the effect on cerebral vessels is so much weaker than that on vessels elsewhere, that the cerebral vessels will be passively dilated whenever epinephrine is distributed uniformly throughout the body. Thus epinephrine, given systemically, will only increase cerebral blood flow, dilate cerebral vessels, and exaggerate any existing tendency to cerebral hemorrhage.[6, 23]

The results of pituitrin and pitressin studies in animals indicate no direct effects upon the cerebral circulation. The effects of angiotonin or hypertension should be interesting but have not yet been demonstrated.

Drugs: Among the vasodilators a large number of drugs may be capable of cerebral-vasodilatation. For example: the nitrites, choline derivatives, the xanthine group (caffeine, theophylline, theobromine), histamine, the opium alkaloids (morphine, codeine, papaverine), the entire group of narcotics and anesthetics, and calcium and potassium ions are included in this large group. Since, however, the intracranial vessels are generally less sensitive to these drugs than the extracranial vessels, a drug concentration high enough to act all over the body would probably bring about a redistribution of the blood at the expense of the brain and a reduction in the cerebral blood flow. Carbon dioxide may be mentioned as an



a good "mixer"

for your cough prescriptions

especially valuable when allergic factor

is suspected or present

Chlor-Trimeton syrup q.s. ad

- taste appeals to young and old
- compatible with commonly prescribed medications

Contains CHLOR-*TRIMETON*® Maleate
(brand of chlorprophenpyridamine maleate), 2 mg. per teaspoonful (4 cc.).

Schering

CHLOR-*TRIMETON* SYRUP

SCHERING

outstanding exception to this, for here the vasomotor center is stimulated while the cerebral blood vessels are dilated.[23]

Shenkin,[25] using the nitrous-oxide method of Kety and Schmidt,[16] has studied the effects of caffeine, papavarine, histamine, and benzedrine upon the cerebral circulation of man. These recent experiments have shown that caffeine produces increased vascular resistance, a reduced cerebral blood flow (15.6 per cent), and little change in cerebral O_2 consumption. Papavarine produced significant vasculature relaxation but reduced the mean arterial pressure so that the cerebral flow was not significantly increased. Benzedrine did not produce any marked effects. Histamine, however, proved to be an excellent cerebral vasodilator, causing an increase in the cerebral blood flow when the general arterial pressure was reduced as much as 15 per cent. Greater reduction in the arterial pressure caused proportional reduction of the cerebral circulation. Furmanski[8] has shown clinically in a series of 25 patients with brain ischemia due to thrombosis, embolism, etc., that 20 responded with definite improvement to intravenous histamine therapy. (Histamine diphosphate 2.75 mg. per 500 cc. isotonic fluid I V over two hours.)

The strongest vasoconstrictor of the cerebral vessels yet demonstrated is that of decreased CO_2 tension. Recent investigations, using the nitrous oxide technique,[13, 27] have shown that aminophylline given intravenously in therapeutic dosage in man produces a constant cerebral vasoconstriction, a significant reduction in cerebral blood flow, and a resultant cerebral anoxia. It has also been shown that dihydroergocornine causes a release of cerebrovascular resistance in patients with essential hypertension and to a lesser extent in normal subjects.[11]

IV. EXPERIMENTAL TECHNIQUES[10]

Changes in pial vessels: Changes are usually observed only in local areas, and this also does not necessarily portray the picture of what is going on in arterioles deep in the cerebral tissue. Also these observations may not always apply to the unanesthetized animal.

Perfusion technique: Here the cerebral flow is maintained by a mechanical pump, and the observations are made under unphysiological conditions.

Measurement of flow by instruments attached to vessels: If measurements are made on one vessel, the others being untied, they may or may not record directional changes correctly. If all other arteries are tied, the specimen becomes unphysiological.

Measurement of blood flow in the intact blood vessel: There are many instruments used, such as thermostromuhrs, etc., and here the many technical variables are difficult to control.

Measurement of blood flow with thermoelectric instruments: Here again are many variables which are difficult to control. The method is not specific and is not quantitative. There are too many sources of error.

Cerebral AV O_2 differences: These have been used as an index of cerebral metabolism. This method does not take into account extra-metabolic factors which may alter the rate of cerebral flow and consequently change the AV O_2 difference. Here again the variables are not brought into control. This method has also been used as a measure of cerebral blood flow, assuming the cerebral metabolism as constant, an assumption which for these purposes is not justifiable.

Plethysmographic method: This method involves the use of a cuff about the neck, at a pressure of 60-80 mm. Hg. in order to stop venous return. Here it is assumed that the CSF is displaced, is proportional to the cerebral flow, and can be measured by the rate of CSF flow through a lumbar needle. According to Gregg and Shipley,[10] the venous outflow from the cranium is not halted by this procedure.

With the exception of the perfusion experiments, all of the preceding procedures have involved making observations from which the magnitude of, or directional changes in blood flow are determined only indirectly. In order that any direct method be considered valid, it is necessary that (1) the available quantity actually measured varies in constant relationship to the unavailable and unknown quantity being measured indirectly; and (2) other variables which change spontaneously or are induced by the procedure do not modify this relationship.[10]

Nitrous oxide method: A short time following the symposium on the cerebral circulation, Kety[16] conceived and subsequently developed a method which has made possible the quantitative measurement of cerebral blood flow and cerebral metabolism in intact man.[13] This is now known as the nitrous oxide method. This method involves the inhalation of a mixture of 15 per cent N_2O, 21 per cent O_2, and 64 per cent N_2. Blood samples are collected simultaneously from an artery and the superior jugular bulb; (1) during the first minute of inhalation, (2) at the end of the first minute, (3) the third minute, (4) the fifth minute, and (5) the tenth minute. An analysis is made of the N_2O content in the blood. Cerebral AV O_2 differences are calculated from the O_2 content in samples of blood taken before inhalation and after five minutes of inhalation. The mean arterial blood pressure is also determined during the procedure. From these measurements the blood flow in cc./100 Gm. brain/min.; the O_2 consumption in cc./100 Gm./min.; and the vascular resistance can be calculated.

The significance of this new method is far reaching, for now it is possible to determine the effects

tested

broad-spectrum therapy

Terramycin®

BRAND OF OXYTETRACYCLINE

established

an agent of choice

in the treatment of a wide range of infections due to gram-positive and gram-negative bacteria, spirochetes, rickettsiae, certain large viruses and protozoa.

clinical advantages known

rapid absorption

wide distribution

prompt response

excellent toleration

Within an hour after oral administration in fasting or non-fasting state, effective serum concentrations of Terramycin may be attained.[1] It is widely distributed in body fluids, organs and tissues and diffuses readily through the placental membrane.[2,3] Immediate evidence of Terramycin's efficacy is often obtained by the rapid return of temperature to normal.[4] Widely used among patients of all ages, this tested broad-spectrum antibiotic is well tolerated.[5,6]

1. Sayer, R. J., et al.: Am. J. M. Sc. *221*:256 (Mar.) 1951.
2. Welch, H.: Ann. New York Acad. Sc. *53*:253 (Sept.) 1950.
3. Werner, C. A., et al.: Proc. Soc. Exper. Biol. & Med. *74*:261 (June) 1950.
4. Wolman, B., et al.: Brit. M. J. *1*:419 (Feb. 23) 1952.
5. Potterfield, T. G., et al.: J. Philadelphia Gen. Hosp. *2*:6 (Jan.) 1951.
6. King, E. Q., et al.: J. A. M. A. *143*:1 (May 6) 1950.

Available in convenient oral, parenteral and ophthalmic preparations.

Pfizer

PFIZER LABORATORIES, *Brooklyn 6, N. Y.*
Division, Chas. Pfizer & Co., Inc.

upon the cerebral blood flow and metabolism of the many drugs, hormones, nervous stimulation, hyperventilation, anoxia, and surgical procedures (i.e., carotid ligation) with a reasonable degree of accuracy.

N_2O method:[20] 1. Values for cerebral blood flow per 100 Gm. of brain, arterio-venous O_2 difference, A-V glucose difference, cerebral O_2 utilization, cerebral glucose utilization, and cerebrovascular resistance are given in 29 observations on 23 patients with cerebral vascular disease. The subjects were either middle aged persons with hypertension and diabetes or patients with a history of a CVA. They were divided into those with normal and those with abnormal mental status.

2. Those patients who had no alteration in their mental status had significantly lower cerebral blood flows, higher AV O_2 differences, and higher cerebrovascular resistances than normal young persons. Cerebral O_2 and cerebral glucose utilizations were normal.

3. The subjects with abnormal mental status resulting from cerebral vascular disease had significantly lower cerebral blood flows, cerebral O_2 and glucose utilizations, and higher cerebrovascular resistances than those with normal mental status or than normal young subjects.

4. It is believed that these groups represent two stages in the natural progression of cerebral vascular disease.

5. Unilateral stellate ganglion block produced no change in cerebral metabolic functions in normals, patients with hypertensive vascular disease, and elderly patients with cerebral thrombosis.

It is logical to assume that the reduction in blood flow is the result of diffuse vascular disease and not hypertension per se, since Kety and colleagues[14] have shown that in uncomplicated hypertension the cerebral blood flow and AV O_2 difference are normal though the cerebrovascular resistance is increased.

The decrease in cerebral metabolism is related to the inability of the brain cells to extract more O_2 and glucose per unit of blood, rather than to the low blood flow itself. This poor cellular function results in some way from the progressive vascular disease. Several possibilities exist, i. e.:

1. Insufficient areas of softening and scarring may have occurred so that part of the blood supply to the brain passes through areas of scar tissue.

2. Marked disturbances in function of uninjured cells may have resulted from the great disorder in total brain function produced by many local lesions.

3. In certain areas metabolism of existing cells may be greatly deranged by reductions in cerebral blood flow to a level incompatible with normal function but not sufficient to cause death.

4. Infection and altered metabolism in other organs may secondarily affect brain metabolism.

Arteriographic demonstration of arterial spasm:[5] By means of comparative arteriograms, they demonstrated arterial spasms (in vessels with interior diameter at least 1.5 mm.) in 12 instances among 10 cases. Vessels involved were internal carotid, anterior cerebral, middle cerebral arteries, and their branches.

The common element in the production of spasm in all cases seemed to be abrupt traction on the arterial wall, i.e. from association with aneurism, hemorrhage (local), edema, tumor, and severe intrinsic lesion of the arterial wall.

Usually spasm was maximal at the lesion but extended several centimeters along adjacent arteries in lesser degree.

V. CEREBRAL ANGIOSPASM

From a clinical standpoint, the physician over a period of years will see and treat a considerable number of patients with cerebral attacks. These attacks, of course, are characterized by a variety of symptoms depending upon the underlying pathology. The symptoms of cerebral hemorrhage and cerebral thrombosis are often quite obvious and the gross pathology quite evident.

There are, however, other types of cerebral attacks of a lesser degree in which the disturbances are of a much shorter duration, transient disturbances with perhaps no residual, in which the gross appearance of the brain after death is less noticeably altered.

It is with these transient episodes that we are here concerned. Even more specifically we are concerned with the question of whether or not cerebral angiospasm, per se, can and does produce these episodes. This may or may not seem a moot point, depending upon one's views. However, considering the fairly widespread acceptance of the idea of cerebral angiospasm, especially prior to World War II, and considering the experimental advances in studying cerebral circulation and the trends of thought since this time, his point is here thought to be worthwhile.

Pickering's excellent paper in 1948 on transient cerebral paralysis[19] describes encephalopathy in acute hypertension, in chronic hypertension, and that in cerebral embolism. He presents 22 cases of cerebral attacks, 11 of which showed transient motor or sensory paralysis of cerebral origin in essential hypertension, and 11 cases of cerebral embolism in mitral stenosis and auricular fibrillation. He points out the close similarity clinically and pathologically between the attacks in the two conditions and suggests a corresponding similarity in the underlying vascular events. In each instance the onset of the attack is due to organic arterial obstruction of sudden development and resolution due to restoration of circulation through dilatation of collateral vessels. The obstruction in one case is due to an embolus and in

use a drug of choice

Erythrocin

TRADE MARK

(Erythromycin, Abbott)

"Erythromycin, given orally, is an effective antibiotic and seems to be an antibiotic of choice, at present, in the treatment of infections due to resistant strains of staphylococci."[1]

HIGHLY-ACTIVE ERYTHROCIN is also effective against streptococci and pneumococci. Less likely to alter normal intestinal flora than most other oral antibiotics. Gastrointestinal disturbances rare, with no serious side effects reported.

AVERAGE ADULT DOSE: 200 mg. every four to six hours. You'll find *Specially-coated* ERYTHROCIN tablets (100 and 200 mg.) in bottles of 25 and 100 at all pharmacies. Abbott

1. Grigsby, M. E., et al., Antibiot. & Chemother., 10:1029, October, 1953.

ALSO FOR CHILDREN: Tasty, Stable *Pediatric* ERYTHROCIN Suspension

1-76 54

the other to thrombus formation. He attributes acute hypertensive episodes to cerebral edema as a result of defective vasoconstriction.

Latham[18] has demonstrated microscopic cerebrovascular lesions in a variety of organic diseases where the large vessels may not appear enlarged, and he points out Biggart's dictum: "The naked eye appearance of the larger vessels is no criterion of the presence or absence of vascular damage."

Kety[13] states that it is possible that reflex cerebrovascular spasm might occur in response to embolism or thrombosis and suggests that in that type of carotid sinus syndrome which produces syncope without a bradycardia or a fall in blood pressure an intense vasoconstriction might be produced, an exaggeration of the normal response. "Indeed, the entire question of cerebrovascular spasm to a degree sufficient to embarrass the cerebral circulation is a problem which requires much further investigation."

Schmidt[23] believes that while the possibility of cerebral angiospasm cannot be denied, the evidence is greatly against it.

Scheinker[21] states in regard to cerebral angiospasm: "Clinically it is not whether angiospasm can and does occur, but whether it is of significance to induce a variety of clinical manifestations. There is no direct or indirect proof that angiospastic phenomena are of paramount significance in migraine, epilepsy, hypertension, multiple sclerosis, etc. That spasm of an individual blood vessel is ever of sufficient intensity and duration to induce permanent damage to the tissue it supplies is questionable. It is to be little doubted that vascular dilatation to an extreme degree, in association with stasis, is by far the most striking and essential feature in such forms of functional vascular disturbances as vasoporosis, vasoparalysis, and vasothrombosis. The vasoparalytic dilatation of a cerebral blood vessel results in a retardation in cerebral blood flow, and there is every indication that the margin of safety of the nerve parenchyma is much smaller than that of any other organ, with regard to volume of blood per unit of time and percentage of O_2 carried by the blood. An extreme vascular dilatation, associated with stasis and retardation or cessation of blood flow, will thus result in an interference of O_2 and CO_2 exchange and affect tissue function."

Scheinker supports the idea of vasoparalytic phenomena possibly preceded by brief angiospasm. Transient clinical symptoms, such as hemiplegia and aphasia, do not rule out small focal areas of tissue destruction.

Clinically it might be said that transient cerebral attacks might well be a warning to the physician to consider other pathology before ascribing them to "angiospasm" per se.

VI. SUMMARY

1. The cerebral circulation is discussed from the standpoint of anatomy (both gross and microscopic), physiology, and recent experimental developments.

2. The subject of cerebral angiospasm is considered.

BIBLIOGRAPHY

1. Batson, D. V.: Fed. Proc. 3:139, 1944.
2. Batty, L. L.; Heyman, A., and Patterson, J. L., Jr.: J.A.M.A., 152:6, 1953.
3. Chorobski, J., and Penfield, W.: Arch. Neurol. & Psychiat. 28:1257, 1932.
4. Cobb, S., and Lennox, W. G.: Fed. Proc. 3:151, 1944.
5. Ecker, H., and Riemenschneider, P. A.: J. Neurosurg. 8:660, 1951.
6. Fog, M.: Arch. Neurol. & Psychiat. 41:109, 1939.
7. Forbes, H. S., and Wolff, H. G.: Arch. Neur. Psychiat. 19:1057, 1928.
8. Furmanski, A. R.: Arch. Neurol. Psychiat. 63:415, 1950.
9. Gibbs, F. A.; Maxwell, H., and Gibbs, E. L.: Arch. Neurol. Psychiat. 57:137, 1947.
10. Gregg, D. E., and Shipley, R. E.: Fed. Proc. 3:144, 1944.
11. Hafkenschiel, J. H.; Crumpton, C. W.; Moyer, J. H., and Jeffers, W. A.: J. Clin. Invest. 29:408, 1950.
12. Humphreys, S. P.: Arch. Neurol. Psychiat. 41(6):1207, 1939.
13. Kety, S. S.: Am. J. of Med. 8:205, 1950.
14. Kety, S. S., et al.: J. Clin. Invest. 27:511, 1948.
15. Kety, S. S., and Schmidt, C. F.: J. Clin. Invest. 27:484, 1948.
16. Kety, S. S., and Schmidt, C. F.: J. Clin. Invest. 27:476, 1948.
17. Kety, S. S.; Polis, B. D.; Nadler, C. S., and Schmidt, C. F.: J. Clin. Invest. 27:500, 1948.
18. Latham, O.: Med. J. Australia 1(7):292, 1938.
19. Pickering, G. W.: J.A.M.A. 137:423, 1948.
20. Scheinberg, P.: Am. J. Med. 8:139, 1950.
21. Scheinker, I. M.: Medical Neuropathology, Thomas Pub. Co., Springfield, Ill., 1951.
22. Schmidt, C. F.: Fed. Proc. 3:131, 1944.
23. Schmidt, C. F.: The cerebral circulation in health and disease, Thomas Pub. Co., Springfield, Ill., 1950.
24. Shenkin, H. A.; Harmel, M. H., and Kety, S. S.: Arch. Neurol. Psychiat. 60(3):240, 1948.
25. Shenkin, H. A.: J. Appl. Physiol. 3:465, 1951.
26. Shenkin, H. A., et al.: J. Neurosurg. 8:38, 1951.
26a. Steegmann, A. T.: Neurology 1:261, 1951.
27. Wechsler, R. L.; Kleiss, L. M., and Kety, S. S.: J. Clin. Invest. 29:297, 1950.

Regional Meeting on Veterans' Care

A series of regional conferences on veterans' medical care will be held during 1954 under the sponsorship of the A.M.A.'s Council on Medical Service. The meeting for this area will be held at Denver on February 21.

These meetings have a fourfold purpose: (1) develop a working liaison with state society committees concerned with veterans' problems; (2) acquaint an increasing number of physicians with A.M.A. policy and the facts regarding veterans' care; (3) learn the local situation regarding this policy in various states, and (4) discuss ways of carrying out the instructions of the House of Delegates which adopted a firm stand on veterans' medical care at its meeting last June.

It is not too early to make hotel reservations for the 95th annual session, Kansas Medical Society, Topeka, May 2-6, 1954.

Upjohn

less-antigenic penicillin:

Cer-O-Cillin

Trademark Reg. U. S. Pat. Off.

POTASSIUM

ACCEPTED COUNCIL ON PHARMACY AND CHEMISTRY AMERICAN DENTAL ASSOCIATION

Available as.

Sterile vials containing 200,000 and 500,000 units Crystalline Penicillin O Potassium.

Bottles of 12 buffered tablets, each containing 100,000 units Crystalline Penicillin O Potassium.

Depo*-Cer-O-Cillin Chloroprocaine for Aqueous Injection in vials containing 1,500,000 units Crystalline Chloroprocaine Penicillin O.

*TRADEMARK, REG. U.S. PAT. OFF.

The Upjohn Company, Kalamazoo, Michigan



ANNOUNCEMENTS

An "extra large" four-day sectional meeting of the American College of Surgeons, featuring clinics and specialty programs, will be held at the Hotel Fontenelle in Omaha, March 1-4. All members of the medical profession are invited to attend.

The Foundation of the American Society of Plastic and Reconstructive Surgery announces an essay contest in two divisions, for those who are residents or who have practiced plastic surgery for no longer than five years and for those who have been active in the specialty for more than five years. Manuscripts will be accepted until July 1, 1954. Complete information may be secured from the Award Committee, c/o Jacques W. Maliniac, M.D., 30 Central Park South, New York, New York.

The Sixth Annual Symposium on Recent Advances in the Study of Venereal Diseases will be held in the auditorium of the Department of Health, Education, and Welfare, Washington, D. C., April 29 and 30. Sessions are open to all physicians interested in participating.

All physicians are invited to attend the Sixth Annual Convention of the International Academy of Proctology to be held at the Palmer House, Chicago, April 8-11. Much of the program has been planned to answer questions of general practitioners, gastroenterologists, and proctologists. There is no fee for attendance. Programs are available from the Executive Office of the Academy, 43-55 Kissena Boulevard, Flushing, New York.

The National Foundation for Infantile Paralysis announces the availability of a limited number of postdoctoral clinical fellowships in physical medicine

UNIVERSITY OF KANSAS SCHOOL OF MEDICINE

Postgraduate Medical Courses

PUBLIC HEALTH AND PREVENTIVE MEDICINE
March 17, 18 & 19

Guest Instructors:
GAYLORD W. ANDERSON, M.D., Mayo Professor and Director, School of Public Health, University of Minnesota School of Medicine, Minneapolis.
RUSSELL J. BLATTNER, M.D., Professor of Pediatrics & Chairman of Department, Baylor University College of Medicine, Houston, Texas.
WILLIAM M. BUSCH, LL.B., Business Manager and Attorney, Kansas State Board of Health, Topeka.
DONALD A. DUKELOW, M.D., Consultant in Health and Fitness, Bureau of Health Education, American Medical Association, Chicago, Ill.
THOMAS R. HOOD, M.D., Executive Secretary, Kansas State Board of Health, Topeka.
HARLAN R. SCHUYLER, M.S., Ph.D., Entomologist; President of Conservation Industries, Kansas City, Kans.
GEORGE M. WHEATLEY, M.D., Vice-President in Charge of Health and Welfare, Metropolitan Life Insurance Company, New York City.
Subjects to Be Discussed:
Rights, Privileges and Immunities of the Local Health Officer.
Statutory Responsibilities and Prerogatives of the Local Health Officer.
Use of the Voluntary Health Agencies by the Local Health Officer.
The Local Health Officer and Problems in Sanitation.
Health Is Everybody's Business.
A Croaking Frog and Three Mad Foxes.
International Control of Disease as It Affects the Local Health Officer.
Health Hazards of Insecticides and Rodenticides.
Toxoplasmosis.
Infectious Hepatitis.
The Physician and Child Health Services.
Needs of Children Today for Hospital and Health Services.
The Physician and the Schools.
Present-Day Concepts of Communicable Disease Control.
Comprehensive Medical Care for Children (a Panel Discussion).
Retrolental Fibroplasia; New Developments in Etiology and Prevention.
Etiologic Factors in Enteric Infections.
Prevention of Recurrent Rheumatic Fever.
Use of Gamma Globulin; Recent Developments in Poliomyelitis.
Encephalitis.
Treatment of Enuresis.
Importance of Tuberculin Skin Testing in Children.

HEMATOLOGY
Laboratory Course
March 11 & 12

University of Kansas Faculty:
KURT R. REISSMANN, M.D., Associate Professor of Medicine and Chairman, Experimental Medicine Section.
SLOAN J. WILSON, M.D., Associate Professor of Medicine and Hematologist to the Medical Center.
Also several staff assistants.
Guest Instructor: MARJORIE SIRRIDGE, M.D.
Subjects to Be Discussed:
Erythropoiesis and Iron Metabolism.
Examination of Blood and Bone Marrow in Red Cell Diseases.
The Anemias.
Leukocyte Formation and Their Relation to Disease.
Benign and Malignant Leukocytic Diseases, Peripheral Blood and Bone Marrow (3 two-hour sessions).
Panel Discussion on Benign Leukocytic Diseases.
Acute and Chronic Leukemia; Diagnosis and Prognosis.
Marrow Megakaryocytes in Purpura.
Panel Discussion on the Malignant Hematologic Diseases.
Demonstration of Hematologic Techniques.

DIAGNOSTIC X-RAY AND FLUOROSCOPY
March 11 to April 15, 1954

All-day program on Thursdays—6 weeks
Enrollment Limited

PHYSICAL DIAGNOSIS AND AUSCULTATION OF THE HEART
March 22, 23, 24 & 25

Guest Instructors:
ERNEST CRAIGE, M.D., University of North Carolina.
J. WILLIS HURST, M.D., Emory University.
GORDON S. MYERS, M.D., Massachusetts General Hospital; and three additional staff members of the Cardiac Laboratory.

It's Convenient to Buy Kanox

THERAPEUTIC OXYGEN AND ANESTHETIC GASES



Oxygen Therapy
Regulators and Equipment
Repair Service for
Regulators, Flowmeters
and Humidifiers

Nitrous Oxide
Ethylene
Carbon Dioxide
Helium
Oxygen
Cyclopropane

KANSAS OXYGEN, Inc.

Phone 5-5551 HUTCHINSON, KANSAS Box 551



Woodcroft Hospital--Pueblo, Colorado

A private hospital for the scientific treatment of neuro-psychiatric disorders, including alcoholism and drug addiction. Beautiful landscaping and home-like surroundings afford a restful atmosphere. Accommodations vary from single rooms with or without bath to rooms en suite, allowing for segregation of guests.

Detailed information furnished on request.

KARL J. WAGGENER, M.D.

and rehabilitation to candidates who wish to become eligible for certification in that field. Fellowships will cover a period of one to three years. Information may be secured from the Division of Professional Education, National Foundation for Infantile Paralysis, 120 Broadway, New York 5, New York.

The 1954 meeting of the American Goiter Association will be held at the Somerset Hotel, Boston, April 29-May 1. The three-day program will consist of papers and discussions dealing with the physiology and diseases of the thyroid gland.

The National Gastroenterological Association announces that the name of its official publication, established in 1934, has been changed from *The Review of Gastroenterology* to *The American Journal of Gastroenterology,* effective with the January issue. The current issue is Volume 21, Number 1.

Physicians are invited to attend the Sixth Congress of the Pan-Pacific Surgical Association to be held in Honolulu, October 7-8, 1954. The scientific program, presented by more than 100 surgeons, will include sessions in all divisions of surgery and related fields. A social program is being developed for the doctors' families. Complete information may be secured from F. J. Pinkerton, M.D., Director General, Pan-Pacific Surgical Association, Suite Seven, Young Building, Honolulu, Hawaii.

Grant to Committee on Careers in Nursing

A March of Dimes grant of $47,690 will assist the Committee on Careers of the National League for Nursing to continue its national program of recruitment of students for schools of professional and practical nursing.

Since 1949, the Foundation has provided financial assistance. The 1954 grant will underwrite the committee's field services program designed to help state and local committees in their efforts to interest young people in nursing careers.

Contributions to the American Medical Education Foundation during the first 11½ months of 1953 totalled $1,047,000. More than 24,500 physicians contributed $847,361 directly to 70 of the nation's 79 approved medical schools.

Patronize JOURNAL advertisers.

THE DALLAS SOUTHERN CLINICAL SOCIETY

Announces

23rd Annual Spring Clinical Conference

March 15, 16, 17, 18, 1954

WILLARD M. ALLEN, M.D., Gynecology, St. Louis
WILLIAM B. BEAN, M.D., Internal Medicine, Iowa City
EDWARD G. BILLINGS, M.D., Psychiatry, Denver
WILLIAM L. BRADFORD, M.D., Pediatrics, Rochester, New York
ALSON E. BRALEY, M.D., Ophthalmology, Iowa City
ORMOND S. CULP, M.D., Urology, Rochester, Minnesota
CLARENCE DENNIS, M.D., Surgery, Brooklyn
THOMAS J. DRY, M.D., Cardiology, Rochester, Minnesota
CHARLES H. EYERMANN, M.D., Allergy, St. Louis
HUGH F. HARE, M.D., Radiology, Los Angeles
A. C. HILDING, M.D., Otolaryngology, Duluth
SAMUEL F. MARSHALL, M.D., Surgery, Boston
PAUL E. McMASTER, M.D., Orthopedic Surgery, Beverly Hills, California
GEORGE T. PACK, M.D., Surgery, New York
JOHN PARKS, M.D., Obstetrics, Washington, D. C.
DOUGLAS H. SPRUNT, M.D., Pathology, Memphis
C. DWIGHT TOWNES, M.D., Ophthalmology, Louisville
WALTER P. WORK, M.D., Otolaryngology, San Francisco

For information address:
Executive Secretary
433 Medical Arts Building
Dallas 1, Texas

ACCIDENT, HOSPITAL AND SICKNESS INSURANCE

For Physicians, Surgeons and Dentists Exclusively

ALL PREMIUMS COME FROM — PHYSICIANS SURGEONS DENTISTS — ALL CLAIMS GO TO

$5,000 accidental death Quarterly $8.00 $25 weekly indemnity, accident and sickness	$15,000 accidental death Quarterly $24.00 $75 weekly indemnity, accident and sickness
$10,000 accidental death Quarterly $16.00 $50 weekly indemnity, accident and sickness	$20,000 accidental death Quarterly $32.00 $100 weekly indemnity, accident and sickness

Cost Has Never Exceeded Amounts Shown

ALSO HOSPITAL INSURANCE

	Single	*Double*	*Triple*	*Quadruple*
60 days in Hospital	5.00 per day	10.00 per day	15.00 per day	20.00 per day
30 days of Nurse at Home	5.00 per day	10.00 per day	15.00 per day	20.00 per day
Laboratory Fees in Hospital	5.00	10.00	15.00	20.00
Operating Room in Hospital	10.00	20.00	30.00	40.00
Anesthetic in Hospital	10.00	20.00	30.00	40.00
X-Ray in Hospital	10.00	20.00	30.00	40.00
Medicines in Hospital	10.00	20.00	30.00	40.00
Ambulance to or from Hospital	10.00	20.00	30.00	40.00
COSTS (Quarterly)				
Adult	2.50	5.00	7.50	10.00
Child to age 19	1.50	3.00	4.50	6.00
Child over age 19	2.50	5.00	7.50	10.00

$4,000,000.00 INVESTED ASSETS

PHYSICIANS CASUALTY ASSOCIATION
PHYSICIANS HEALTH ASSOCIATION

$19,500,000.00 PAID FOR CLAIMS

51 years under the same management

400 First National Bank Building Omaha 2, Nebraska

$200,000.00 deposited with State of Nebraska for protection of our members

Pure as sunlight



Research on Capillary Membranes

Research on the capillary membranes which help regulate the volume of circulating blood and the blood pressure and influence the abnormal collection of fluid in heart failure and other conditions is being conducted by Dr. John R. Pappenheimer at Harvard Medical School.

Dr. Pappenheimer, an investigator of the American Heart Association, has been studying the processes by which molecules move through the membrane-like walls of the minute blood vessels. These membranes are closely knit and prevent the passage of blood cells and large protein molecules, but smaller molecules of essential chemical substances shuttle through them between the blood stream and the fluid bathing body cells. The rate of movement of these substances by filtration and diffusion influences the supply of nutriment, the removal of waste products, and the balance of fluid vital to health.

The eventual goal of the study is to develop means of counteracting abnormal body processes, thus preventing or controlling disease.

The Motion of the Heart

A book commissioned by the American Heart Association and designed for a lay audience, "The Motion of the Heart," was published by Harper and Brothers on February 3. It describes the methods, objectives, and accomplishments of cardiovascular research. Mr. Blake Cabot wrote the book, which was reviewed before publication by Doctors Robert L. King, H. M. Marvin, Louis N. Katz, Howard B. Sprague, and Irving S. Wright.

Medical Gift to Jungle Hospital

A shipment of 29 tons of pharmaceuticals, more than 100,000 bottles of medicine, has been dispatched by Eli Lilly International Corporation to the jungle hospital of Dr. Albert Schweitzer, winner of the 1952 Nobel peace prize. The hospital is in French Equatorial Africa.

Forrest Teel, president of Lilly International, said, "Lilly International is happy to supply medicines to Dr. Schweitzer. Perhaps no other living physician better represents the devotion of the family doctor to human brotherhood."

Dr. Schweitzer has 245 lepers and 230 other patients in his hospital. He is now busy supervising the construction of buildings financed by the $33,840 Nobel prize.

CLASSIFIED ADVERTISEMENTS

FOR SALE OR RENT—Fully equipped 3-room office, ground floor, instruments and medicines. Town of 800. Nearest competition 20 and 35 miles. Write the Journal 4-54.

FOR SALE—Office equipment, Picker x-ray, steel examining room set, examination chair, cabinets, lamps, fans, Alpine lamp, heat lamp, diathermy, maple reception furniture, oak office furniture, safe, instruments, microscope, complete well equipped office. Reasonable price. Optional—will rent eight-room brick office or sell on reasonable terms. Good location, county seat, large territory, first class city-owned hospital, surgeon needed, good will free. Retiring. Write the Journal 5-54.

WANTED TO BUY—a good used optical trial case. In reply quote price. Write the Journal 16-53.

WANTED—Registered medical technician, male or female, to work in a clinic of 12 doctors and in a midwestern city of 100,000 population. Excellent working conditions, no night calls. Write the Journal 2-54.

FOR SALE—Spencer monocular microscope, like new. Two eyepieces 10x and 6x; three objective lenses 10x, 44x and 95x oil immersion; mechanical stage: substage light condenser and individual lamp; carrying case. Excellent for medical student or physician's laboratory. Price $225. Write the Journal 3-54.

THE MEDICAL PROTECTIVE COMPANY
FORT WAYNE, INDIANA
PROFESSIONAL PROTECTION EXCLUSIVELY SINCE 1899
specialized service assures "know-how"
TOPEKA Office:
J. E. McCurdy, Rep.,
1035 Randolph Avenue,
Telephone 2-3027

Everything for the Laboratory
SOUTHWEST SCIENTIFIC CORPORATION
LABORATORY SUPPLIES AND EQUIPMENT
122 South St. Francis Street
Phone 2-0582 Wichita, Kansas

Since its introduction over four years ago, Chloromycetin has been used by physicians in practically every country of the world. More than 11,000,000 patients have been treated with this important antibiotic—

world's outstanding therapeutic agents.



OMPANY DETROIT 32, MICHIGAN

TABLE OF CONTENTS

MARCH, 1954

Original Articles

Fractures of the Anterior Tibial Spine—L. O. Litton, M.D., Kansas City, Kansas . . . 117

Complications Following Mitral Commissurotomy with Special Reference to Auricular Fibrillation—E. Grey Dimond, M.D., and Everton Marques dos Santos, M.D., Kansas City, Kansas 120

Experimental Production of the "Dry Field" in the Right Auricle and Ventricle—C. S. Stotts, M.D., and Stanley R. Friesen, M.D., Kansas City, Kansas 123

The Low Salt Syndrome in Children during Hot Weather—Mary E. Evans, M.D., Kansas City, Kansas 125

The Use of Trypsin in Rheumatoid Arthritis: A Clinical Study—William H. Goodson, Jr., M.D., Donald L. Rose, M.D., and William S. Alyea, M.D., Kansas City, Kansas . . 129

A Twenty-Four Year Review of Cesarean Sections in Kansas University Hospitals—J. C. Schroll, M.D., and L. A. Calkins, M.D., Kansas City, Kansas 131

Editorials

Eighth Annual K.U. Issue 139

Progress at the Medical School 139

Kansas Medical Day 140

Miscellaneous

Parotid Tumors—Tumor Conference . . . 144

The Pathogenesis of Hypochromic Microcytic Anemia—Senior Thesis 166

Entered as second-class matter at the post office at Fulton, Missouri, June 19, 1953, under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of October 3, 1917, authorized on July 2, 1918. Office of Publication: 1201-05 Bluff Street, Fulton, Missouri.

THE JOURNAL OF THE KANSAS MEDICAL SOCIETY

Editorial Board

Orville R. Clark, M.D., *Editor,* Topeka
John W. Cavanaugh, M.D., Topeka
David E. Gray, M.D., Topeka
Richard Greer, M.D., Topeka
Dwight Lawson, M.D., Topeka

Associate Editors

Donald P. Trees, M.D., Wichita
Glen R. Shepherd, M.D., Kansas City

Information

Ownership: The Journal is a non-profit publication owned and published monthly by the Kansas Medical Society.

Subscription: A year's subscription to the Journal is included in membership in the Kansas Medical Society, with $2.00 of each member's dues apportioned to the Journal. Rates to others, except in foreign countries, $4.00 per year or 60c per copy.

Material: Scientific articles, editorials, and data of general interest are invited from all members. Articles are to be submitted on condition that they are contributed solely to this publication. A right is reserved to reject any material deemed unsatisfactory.

Manuscripts: Only manuscripts that are typewritten on one side, double spaced, and original copies can be accepted. Manuscripts will be returned upon request.

Advertising: All advertising contracts, and all copy from advertisers under contract are subject to approval of the editorial board. Copy should be received by the 20th of the month immediately preceding the month of publication.

Non-Responsibility: Although an effort is made to publish only accurate articles and legitimate advertisements. the Journal denies legal responsibility for any statements, opinions, or advertisements appearing under the names of contributors or concerns.

Pauline Farrell, Managing Editor
and Advertising Manager

Oliver E. Ebel, Business Manager

THE KANSAS MEDICAL SOCIETY

President.....Lucien R. Pyle, M.D., Topeka
Past President....Warren F. Bernstorf, M.D., Winfield
President-Elect.....Murray C. Eddy, M.D., Hays
First Vice-President..John M. Porter, M.D., Concordia
Second Vice-President..Clyde W. Miller, M.D., Wichita
Constitutional Secretary..Dale D. Vermillion, M.D., Goodland
Treasurer.....John L. Lattimore, M.D., Topeka
AMA Delegate 1953-1954, Laurence S. Nelson, M.D., Salina
AMA Delegate 1954-1955, George F. Gsell, M.D., Wichita

Councilors and Councilor Districts

First District.....W. L. Anderson, M.D., Atchison
Second District .....H. P. Jones, M.D., Lawrence
Third District....Charles E. Vestle, M.D., Humboldt
Fourth District.....F. C. Taggart, M.D., Topeka
Fifth District.....Harold M. Glover, M.D., Newton
Sixth District.....Norton L. Francis, M.D., Wichita
Seventh District..Severt A. Anderson, M.D., Clay Center
Eighth District.....A. E. Rueb, M.D., Salina
Ninth District.....James L. Jenson, M.D., Colby
Tenth District.....L. W. Reynolds, M.D., Hays
Eleventh District.....C. V. Black, M.D., Pratt
Twelfth District.....R. G. Klein, M.D., Dodge City

Oliver E. Ebel, Executive Secretary

Rueben M. Dalbec, Executive Assistant

Executive Office: 315 West Fourth Street, Topeka

THE JOURNAL
of the
KANSAS MEDICAL SOCIETY

Copyright, 1954, by The Kansas Medical Society

Volume LV MARCH, 1954 No. 3

Fractures of the Anterior Tibial Spine

L. O. Litton, M.D.
Kansas City, Kansas

Fractures of the anterior tibial spine are limited almost entirely to adolescence. It is not a common fracture, and the reason for its infrequency lies in the position of the anterior spine, which is inaccessible to direct trauma.

ANATOMY OF THE KNEE

To understand this fracture better, a review of the anatomy of the knee is necessary.[7, 8] The tibia at its articulation with the knee is composed of two surfaces, the medial and lateral condyles. These condyles are separated by a roughened, non-articulating area. The raised portion in the center of this area is the tibial spine or intercondyloid eminence. Situated on the tibial spine there are two flattened surfaces known as the anterior and posterior intercondyloid fossae.

The attachment of the anterior cruciate ligament is described differently by various anatomists. Blaisdell[1] found it attached to the anterior fossa blending with the lateral meniscus and passing obliquely upward, backward, and outward and inserting into the medial border of the lateral femoral condyle. Anderson[7] found, in 9 of 16 dissected cadavers, the tibial attachment of the anterior cruciate ligament was the anterior fossa and did not touch the anterior tubercle. In the remaining specimens there was some attachment to the tubercle. The anterior cruciate ligament resists forward displacement of the tibia on the femur and medial rotation of the tibia. In addition to the anterior spine serving as the attachment of the anterior cruciate ligament, it may limit side to side motion of the femur on the tibia.

MECHANISM OF INJURY

Isolated injuries to the anterior tibial spine are almost always the result of an abnormal stress effected on it by the anterior cruciate ligament. Injuries which in adults would cause rupture of the anterior cruciate ligament will result in avulsion of the anterior tibial spine in children. Callender's theory that "bone gives away before fibrous tissue" appears to hold here. This is particularly true in children where the anterior cruciate ligament is stronger than the soft cartilaginous anterior spine of the growing tibia.

That this fracture is produced by the same forces which cause injury to the anterior cruciate ligament was pointed out by Blaisdell.[1] The most common mechanism of injury is a fall or blow against the femur with the knee flexed and the tibia fixed. Another manner in which the anterior tibial spine may be avulsed is a forceable medial rotation of the tibia with the knee in extension. The anterior cruciate ligament avulses the tibial spine in attempting to resist this motion.

It is illogical to assume that rotational injury alone will result in the shearing off of the spine by the medial femoral condyle. If this were to occur, there must be a tear of the collateral ligament. One would also expect extensive damage to the articular surface in addition to the fracture of the spine. This has not been our experience. The only pathology found at open reduction is a cavity in the head of the tibia from which the anterior spine has been avulsed. The fragment is still attached to the ligament and has been retracted upward by the recoil of the ligament.

It is of interest that two of the three cases presented at this time were the result of bicycle accidents. Three of the five cases presented by Bradford et al.[9] were following bicycle accidents, and Christopher's case also was caused by a similar injury.

Department of Orthopedics, University of Kansas School of Medicine.

DIAGNOSIS

The diagnosis of such a fracture is not always easy. An accurate description of the mechanism of injury is usually difficult to obtain because of the age of the patient involved. Certainly a history of forceable medial rotation with the leg in extension, or a blow on the femur with the knee flexed and

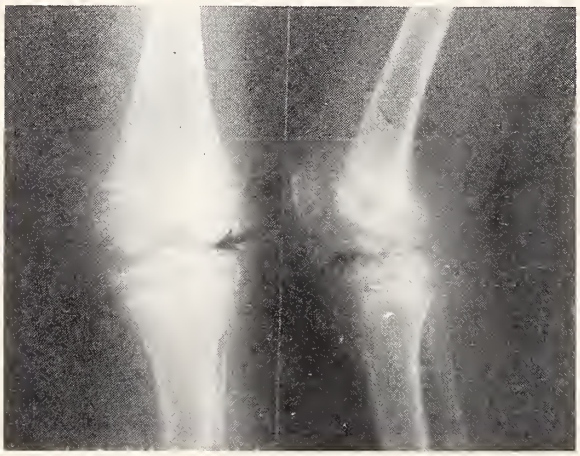


Figure 1

tibia fixed, should make one suspicious of this fracture. There is always an immediate swelling of the knee caused by bleeding into the joint. There is an inability to extend the knee completely. Part of this may be due to the actual locking of the joint by the bone fragment, and part may be due to the loss of the anterior cruciate ligament in the so called "compulsory lateral rotary movement" of the tibia on the femur.

If the examination is done before pain and muscle spasm have occurred, it may be possible to demonstrate abnormal motion of the tibia on the femur. X-ray examination usually reveals the fracture as indicated in Figure 1. This shows the displacement of the fragment into the joint. The size of the fragment varies somewhat, occasionally appearing quite small due to the large amount of unossified cartilage present.

TREATMENT

Most standard orthopedic textbooks recommend closed reduction of this fracture as the treatment of choice. This reduction is accomplished by forcing the leg into full extension. Watson-Jones,[6] Clarke,[2] et al. report excellent results following this procedure. Certainly fractures of the anterior tibial spine with no displacement or with minimal displacement may be treated in this manner, and satisfactory results should be anticipated. Roth,[3] MacAusland[5] and Lee,[4] however, have all shown the impracticality of this method in cases in which displacement of the fragments has occurred. They point out that the anterior cruciate ligament is tight when in extension and hence lifts the fragment from its tibial site when the leg is forced into extension. We found no instance where the meniscus blocked the reduction as described by Roth.[3]

A median parapatellar incision is used for exposure with the distal end of the incision extending well down the tibia. The joint is opened in layers and inspected. The avulsed fragment is then easily replaced. Roth[3] states that fixation is not usually necessary. MacAusland[5] uses a screw for fixation. Lee[4] prefers suturing the fragment into place.

Our method[10] is similar to Lee's except for minor variants. Two drill holes are made on each side of the tibial tubercle and extend upward into the lateral borders of the fracture site. These holes should be on the outside edges of the fracture so that when the fragment is pulled into its bed it will not be held up by the suture. A heavy silk suture is then placed through one of the drill holes and up over the fragment or through it, depending on the size of the fragment. This suture is then pulled down through the other drill hole and through the skin and tied



Figure 2

over a button. Figure 2 shows the position of the pull-out suture and button. The wound is closed, and a cast from the toes to the groin is applied. At the end of about six weeks, the cast and pull-out suture is removed. Quadriceps exercises, of course, are in-

sisted upon during the period of immobilization. Figure 3 demonstrates the healed fracture about eight weeks after operation.

CASE REPORTS

Case 1. M. W., 9-year-old white female. About two weeks prior to admission, this patient was pushed from the bicycle she was riding and fell. She had immediate severe pain with swelling of the left knee. There was marked limitation of motion in extension at that time. There was a great deal of pain associated with attempted weight bearing and motion. X-ray showed an avulsion of the tibial spine. Closed reduction was attempted by placing the knee in complete extension. Check x-rays revealed inadequate reduction. On September 15, 1953, an arthrotomy of the left knee and reduction of the fracture was done by Dr. J. B. Weaver in the manner described above. The postoperative course was uneventful, and the pull-out sutures were removed in about six weeks. At present the patient has a painless knee with full range of motion.

Case 2, J. D., 9-year-old white female. About 10 days prior to admission, the patient fell from her bicycle and injured her left knee. Movement of the knee greatly aggravated the pain, and there was restriction of motion. There was a great deal of swelling of the knee. The knee was placed in as much extension as possible in a long leg cast. Check x-rays revealed some increase in displacement of the fragment. On August 26, 1953, an arthrotomy of the left knee was done by Dr. J. B. Weaver, and the fragment was replaced in the manner described above. Pull-out sutures were removed in about six weeks, and the patient now has a stable knee with full range of painless motion.

Case 3, J. L., 8-year-old white female. The day of admission the patient was pushed from behind and fell to the ground. She had immediate severe pain in the left knee with limitation of extension. X-rays revealed a displaced fracture of the anterior tibial spine. On September 16, 1953, an open reduction was done, and the fragment was replaced in the manner described above except that No. 30 wire was used as the pull-out suture. This wire broke in the subcutaneous region during the period of immobilization and has been left in. This will probably have to be removed at a later date. The fracture has healed, and to date the patient is ambulatory with full flexion and full extension and has no instability or pain.

SUMMARY

1. Fractures of the anterior tibial spine occur most commonly in children.

2. This fracture is the result of an abnormal stress effected on the anterior tibial spine region by the anterior cruciate ligament.

3. Open reduction is the preferred method of treatment in cases with displacement of the fragment.

4. A new method is described to fix the fragment into its tibial site.

5. Three cases are presented to illustrate points in diagnosis and treatment.

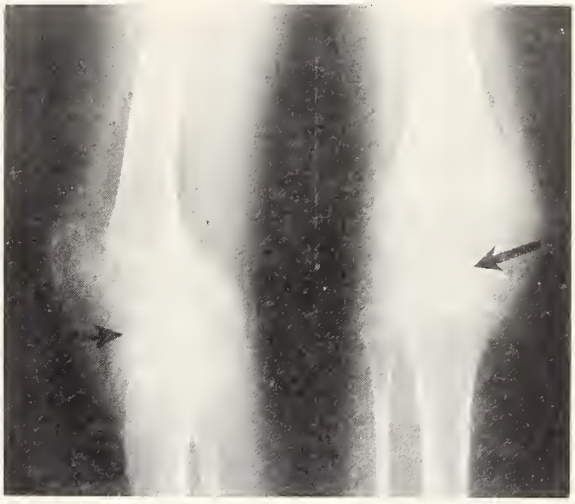


Figure 3

BIBLIOGRAPHY

1. Blaisdell, F. E., Sr.: Fracture of the tibial spine: experimental study, Arch. Surg. 5:561-577, 1922.
2. Clarke, H. O.; Fripp, A. T., and Stamm, T. T.: Discussion on fracture of the tibia involving knee-joint, Proc. Roy. Soc. Med. 28:1035-1050, 1934.
3. Roth, P. B.: Fracture of spine of tibia, J. Bone & Joint Surg. 10:509-518, 1928.
4. Lee, H. G.: Avulsion fracture of tibial attachments of crucial ligaments: treatment by operative reduction, J. Bone & Joint Surg. 19:460-468, 1937.
5. MacAusland, W. R.: Reimplantation of tibial spine in avulsion fracture of anterior crucial ligament, Am. J. Surg. 57:138, 1942.
6. Watson-Jones: Fractures and their treatment. 3rd edition.
7. Anderson, E. R.: Fracture of the spine of the tibia, Minnesota Medicine 23:674-676, 1940.
8. Grant, E.: Method of anatomy. 4th edition.
9. Bradford, C. H.; Adams, R. W., and Magill, H. K.: Fractures of the tibial spines, New England Journal of Medicine 241:137-142, 1949.
10. Weaver, J. B.: Personal communication.

No matter what looms ahead, if you can eat today, enjoy the sunlight today, mix good cheer with friends today, enjoy it and bless God for it. Do not look back on happiness—or dream of it in the future. You are only sure of today; do not let yourself be cheated out of it.

—Henry Ward Beecher

Complications Following Mitral Commissurotomy with Special Reference to Auricular Fibrillation

E. Grey Dimond, M.D., and Everton Marques dos Santos, M.D.

Kansas City, Kansas

We have observed a complication occurring sufficiently often in the postoperative period of mitral commissurotomy to warrant comment. This problem in postoperative management relates to auricular fibrillation.

Recently a paper appeared concerning the development and management of arrhythmias during finger fracture mitral valvuloplasty.[1] Although these acute arrhythmias are disturbing, it is surprising how often they terminate spontaneously. They seldom interfere with the completion of the operative procedure. The arrhythmias that develop either immediately postoperative or in the first few recovery days present a new problem for the clinician, one which seems unique in this type of surgery.

We have selected for mitral commissurotomy 52 cases in the past 21 months. Somewhat to our surprise, the arrhythmia which presented a problem was the occurrence of auricular fibrillation in the postoperative period in patients who had had normal sinus rhythm prior to the valve repair.

Of the 52 patients, 28 had normal sinus rhythm prior to the operation; 8 of these 28 patients developed auricular fibrillation within the first week after the operation.

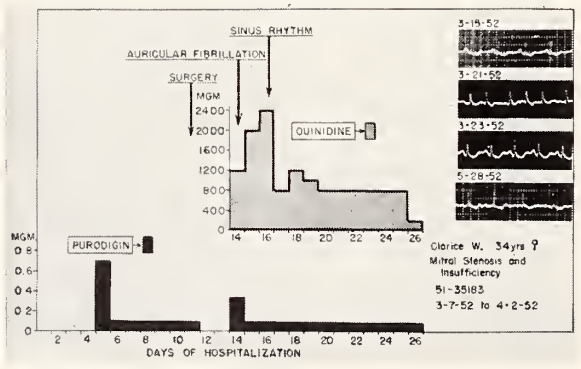


Figure 1.

CASE REPORTS

Case 1, C. W., white female, age 34, KUMC No. 51-35183. Mitral stenosis with minimal insufficiency. Hemoptysis one time. Paroxysmal dyspnea several times. Episodes of palpitation associated with dyspnea lasting one to two hours, starting abruptly and stopping suddenly. Normal sinus rhythm. Operated upon March 19, 1952. On March 21, patient suddenly became weak and began to perspire. Apical rate was 140 and radial rate 68. EKG confirmed auricular

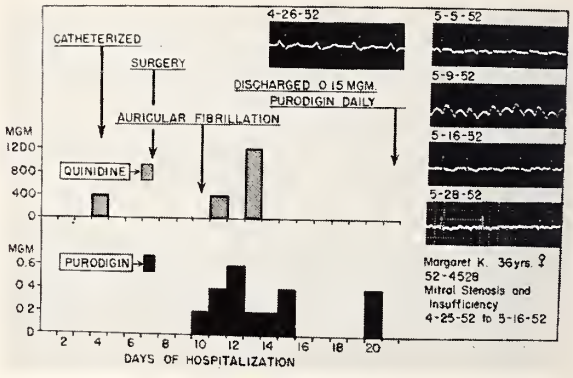


Figure 2.

fibrillation. The subsequent course and management is diagrammed in Figure 1. Biopsy of the auricular appendage revealed chronic endocarditis, mild pericarditis (rheumatic fever), and auricular hypertrophy.

Case 2, M. K., white female, age 36, KUMC No. 52-4528. Mitral stenosis. Shortness of breath on exertion for the past eight years. Normal sinus rhythm. Underwent mitral commissurotomy on May 2, 1952. On May 5 the heart rhythm was found to be irregular with a rate of 160, and the EKG revealed an impure flutter. The attempt at conversion is diagrammed in Figure 2. The patient insisted upon leaving the hospital and was released without further adequate attempt at conversion. Biopsy of the auricular appendage showed hypertrophy of auricular muscle. No evidence of activity.

Case 3, M. S., white female, age 41, KUMC No. 52-7402. Mitral stenosis. Shortness of breath in the last five years. Occasional episodes of palpitation unrelieved by Pronestyl or digitalis. Normal sinus rhythm. Operated upon June 27, 1952. On the day following surgery it was noted that the patient was fibrillating. Conversion was achieved, but auricular fibrillation recurred two times before the normal

From the Department of Medicine, University of Kansas School of Medicine.

sinus rhythm could be maintained. The management of this patient is summarized on Figure 3. Biopsy of the auricular appendage revealed focal myocardial hypertrophy, no evidence of activity.

Case 4, I. H., white female, age 41, KUMC No. 52-13317. Mitral stenosis with minimal aortic involvement. Shortness of breath in the last year. Occasional ankle edema. Normal sinus rhythm. On November 24, 1952, the patient had a mitral finger fracture valvuloplasty. Fibrillation occurred on November 27. The subsequent course and management is shown on Figure 4. The biopsy of the amputated portion of the auricular appendage revealed hypertrophy of the auricular appendage, chronic endocarditis, and the presence of atypical Aschoff bodies.

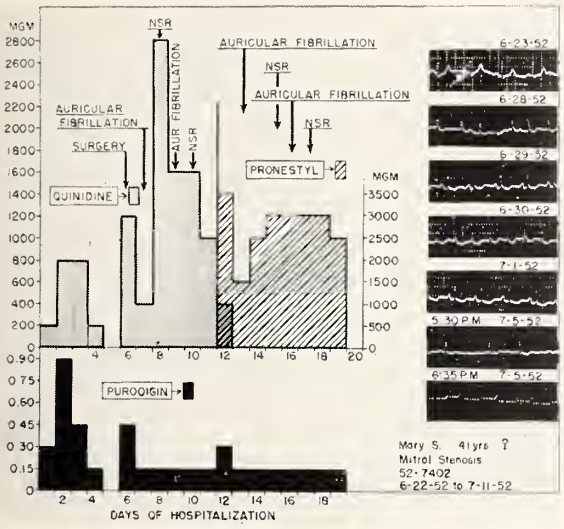


Figure 3.

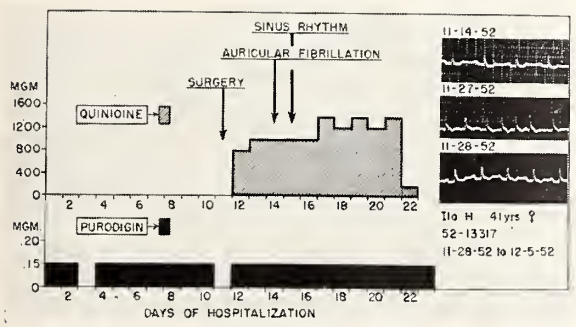


Figure 4.

Case 5, G. L., white male, age 38, KUMC No. 52-13393. Mitral stenosis and aortic stenosis. Came in for "heart operation" because he couldn't get a full breath. Normal sinus rhythm. On November 26, auricular fibrillation developed and was the cause of the prolonged hospitalization of this patient, requiring his further readmission two days after being discharged. The management of this patient's arrhythmia is outlined in Figure 5. Biopsy of the auricular appendage revealed hypertrophy of the auricular appendage, no evidence of activity.

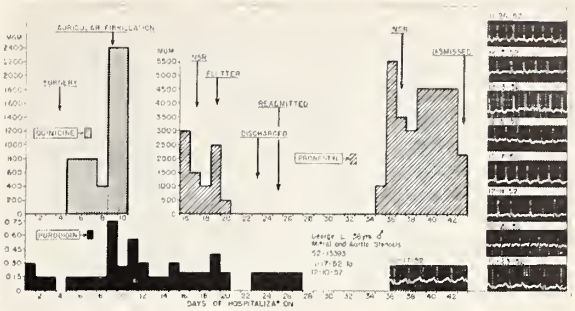


Figure 5.

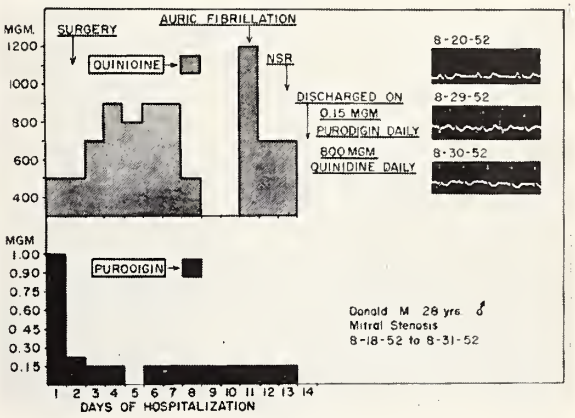


Figure 6.

Case 6, D. M., white male, age 28, KUMC No. 52-7314. Mitral stenosis. Shortness of breath on exertion for one year. Normal sinus rhythm. Surgery deferred two times because of question of rheumatic activity. It finally was done on August 20, 1952. On August 29, the patient was found to be fibrillating. The summary of medication and subsequent course is shown in Figure 6. Biopsy of the auricular appendage revealed evidences of mild chronic pancarditis.

Case 7, J. M., white female, age 27, KUMC No. 53-5850. Pure mitral stenosis discovered nine months ago during pregnancy. The patient had minimal dyspnea at that time, though a cesarean section for placenta previa was done without difficulty. Patient had shortness of breath at exertion on admission here. Mitral commissurotomy was done on May 11, 1953. The next day the patient was found to be fibrillating. Because of nausea, quinidine was not given, but she received additional doses of digitalis. On May 18, 1953, the patient spontaneously reverted to sinus rhythm. Histological pathology reported mild chronic endocarditis.

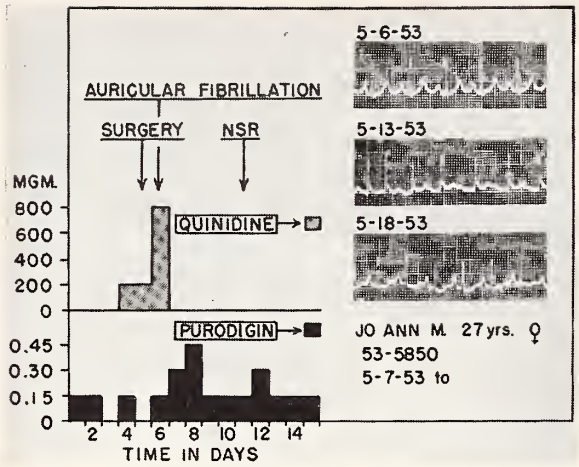


Figure 7.

Case 8, E. C., white female, age 53, KUMC No. 53-5829. Mitral stenosis and insufficiency, aortic stenosis, and minimal regurgitation. History of repeated emboli and paroxysmal nocturnal dyspnea. Mitral

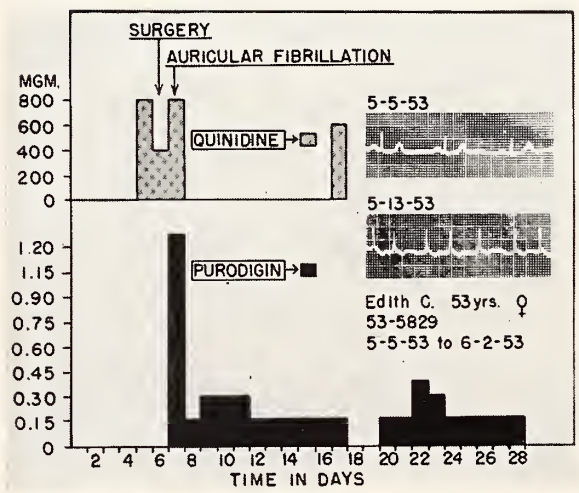


Figure 8.

commissurotomy was done on May 11, 1953, with repair of the valve; regurgitant jet disappeared after valvuloplasty. The next day the patient was found to be fibrillating. Because of nausea, quinidine was not given. One week later patient developed severe postoperative psychosis lasting almost three weeks. Because of this, attempt was made for conversion of auricular fibrillation at the time of this hospitalization. Histological pathology reported hypertrophy of the auricular appendage and mild chronic pericarditis.

DISCUSSION AND MANAGEMENT

Is there any risk in the occurrence of fibrillation in patients after mitral commissurotomy? A principal danger would seem to be the formation of a thrombus in the recently manipulated auricle and embolism. In this group of eight patients, no embolization was recognized clinically; no deaths occurred.

The sudden onset of auricular fibrillation in a patient recently operated upon is cause for considerable concern. The danger from thrombotic complications would seem to be considerably increased in these patients. The recent trauma to the auricle, the inevitable dehydration in a recently operated upon patient, the reduction in cardiac output, and the ineffective contractions of the auricle would set the stage for the serious thrombo-embolic disease. The reduction in cardiac output and possible pulmonary congestion may delay convalescence and favor pulmonary complications.

In these eight patients, a normal sinus rhythm was established in three with quinidine, in two with Pronestyl, and spontaneously in one. In the remaining two patients, our attempt at conversion was inadequate; either insufficient amounts of quinidine and Pronestyl were used or, as in Case 8, no attempt was made during hospitalization because of a severe postoperative psychosis.

SUMMARY OF THE EIGHT CASES (TABLE I)

Name	*Sex*	*Age*	*Development of Arrhyth.*	*Control of Arrhythmia*	*Activity Path.**	*Total Quinid.*	*Medication Prones.*
C.W.	F	34	2 days P.O.	2 days	Yes	4.53 gm	No
M.K.	F	36	3 days P.O.	No	No	0.53 gm	No
M.S.	F	41	1 day P.O.	1 day	No	3.66 gm	22 gm
I.H.	F	41	1 day P.O.	1 day	Yes	3.73 gm	No
G.L.	M	38	6 hours P.O.	27 days	No	2.53 gm	41.75 gm
D.M.	M	28	9 days P.O.	2 days	No	9.2 gm	No
J.M.	F	27	1 day P.O.	7 days	No	+	—
E.C.	F	55	1 day P.O.	No	No	—	—

* Evidence of activity as interpreted from the appendage by the pathologist.

The dose of quinidine and/or Pronestyl required for conversion is charted in Table 1.

The arrhythmia may be recurrent and persistent. For example, Case 3 relapsed into auricular fibrillation three times, and normal sinus rhythm was established three times before the normal rhythm could be maintained.

What is the cause of the arrhythmia? Various factors may be responsible. Of first importance may be the local trauma resulting from the manipulation of the auricle and from the sutures during the surgical procedure. The auricular appendage is clamped, slightly crushed by the Harken or Satinsky clamp, and purse string sutures are placed and rather vigorously pulled; the auricular appendage is amputated and tight sutures are placed. This manipulation is well tolerated, and serious arrhythmias are not common during this period. However, considerable edema and necrosis may occur in the postoperative period and may be responsible for the arrhythmia which we have observed.

A second cause to be considered is the possible reactivation of rheumatic activity in the amputated appendage.[2, 3] There was no correlation (Table 1). Three cases showed evidence of pathological activity; five did not.

The same lack of correlation frequently exists in regard to laboratory evidence of activity, including antistreptolysin titre, diphenylamine test, and heparin tolerance tests. The significance of the pathologist's finding of "activity" needs to be evaluated. Certainly there is little correlation between the pathologist's report and the clinician's observation. We have repeatedly and unsuccessfully attempted to correlate tests for rheumatic activity, pathological evidence of activity in the appendage, and the clinical course.

CONCLUSION

1. An unusual complication may occur in the postoperative period of mitral commissurotomy. In the immediate postoperative period, within the first week, auricular fibrillation may occur in patients who have previously had normal sinus rhythms. A normal sinus rhythm can usually be restored promptly.

2. In 28 patients undergoing mitral valvulotomy and with a normal sinus rhythm prior to operation, auricular fibrillation occurred in patients within the first week.

3. The auricular fibrillation was stopped successfully in six of the eight patients.

4. No correlation between occult activity in the amputated appendage and the occurrence of the arrhythmia was noted.

5. The arrhythmia may be associated with trauma and edema secondary to clamping, sutures, and amputation of the auricular appendage.

BIBLIOGRAPHY

1. Speigl, R. J., et al.: Clinical observations in patients undergoing finger fracture mitral valvuloplasty, Amer. Ht. J., Vol. 12:631, 1952.

2. Janton, Glover, O'Neill: Results of the surgical treatment of mitral stenosis: analysis of 100 consecutive cases, June 8-9, 1951.

3. Soloff, Zatuchni, Janton, O'Neill, and Glover: Reactivation of rheumatic fever following mitral commissurotomy, 26th Scientific Sessions, American Heart Association, April 9-12, 1952.

Experimental Production of the "Dry Field" in The Right Auricle and Ventricle

C. S. Stotts, M.D., and Stanley R. Friesen, M.D.

Kansas City, Kansas

The field of cardiac surgery has expanded greatly in the past few years. Recently there has been much interest in the surgical repair of atrial septum defects. New avenues of approach to and methods of manipulation in the right auricle are constantly being discovered.[1] Hypothermia combined with ganglioplegic hypotensive anesthesia has made it possible to clamp the superior and inferior vena cavae of dogs for as long as 20 minutes without apparent damage.[1] With the use of the "atrial well," the surgeon can operate in the right auricle without haste.[2] The wall of the right auricle as well as separate sections of the wall are used as patches to occlude interatrial septal defects. Prostheses of plastic and polythene are sutured and clamped into positions occluding these septal defects.[3]

This paper is a preliminary report of investigations of methods of approach to the heart and intracardiac manipulation. Our prime goal in this series of experiments has been to develop a "dry field" method of intracardiac manipulation. The advantages of surgery done under direct vision, contrasted to that done through several centimeters of blood or by blind manipulation from the outside of the heart, are obvious. Clamping the superior vena cava, inferior vena cava, and the azygos vein will exclude all the incoming venous flow to the heart except the small coronary flow. When this has been done, the auricle can be opened, the remaining blood removed by suction, and an excellent view of the right auricle obtained. Also the right ventricle can be opened and explored under direct vision.

The authors had found by previous experimentation that five minutes occlusion of the cavae by such a method was the upper limit of safety. Andreasen and Watson have reported two series of dogs in which the vena cavae were clamped with the azygos ligated. They found their dogs died, starting at five minutes, and none survived over eight minutes. In a second series of 13 dogs, the clamps on the cavae were released after a five-minute period of occlusion. Five dogs lived without sequelae; five dogs died, and

From the Department of Surgery, University of Kansas School of Medicine.

the remaining three dogs had severe neurological disturbances.[4]

Cohen, Hammerstrom, Spellman, Varco,[5] and Lillehei have completely occluded the caval circulation in a series of dogs for periods of three, four, five, six, and seven minutes. There was no immediate mortality or morbidity in the three-and four-minute series. One dog out of five in the five-minute class developed spasticity, blindness, and nystagmus.[5]

Bigelow and others have used hypothermia to reduce oxygen consumption, cardiac output, blood pressure, and heart rate. It has been found possible to completely exclude the heart from the circulation at 20°C. for as much as 15 minutes with survival.[6]

To investigate practical limits of "dry field" procedure the authors tested it on dogs. Intravenous nembutal with endotracheal positive pressure oxygen anesthesia was used. A right thoracotomy was performed through the fifth interspace. The azygos was ligated with silk just distal to its junction with the right auricle. The superior and inferior vena cavae were carefully isolated just distal to the junction with the right auricle, and lengths of umbilical tape were placed about them. These tapes were used to occlude the vessels with the aid of a hemostat clamped to the snugged up tape. The pericardium was opened, exposing the right auricle and ventricle. Stay sutures of silk were placed in the auricular wall. At this time the vena cavae were occluded and the auricle incised between the stay sutures. This exposed the right auricle and its communicating passages. The use of suction was occasionally necessary to maintain a completely dry field. To close the auricle the stay sutures were used to approximate the incision, and a Satinski clamp was applied to the incision. The tapes were then released.

Ten dogs were operated upon with the above technique. The time limit for occluding both the superior and inferior vena cava was one and one-half minutes. This occlusion was repeated 10 times in each dog, giving a total time for intracardiac manipulation of 15 minutes. All dogs survived, and in none was there any pathological sequelae other than that normally following thoracotomy in dogs. These dogs have been observed postoperatively for as long as seven months.

Elective autopsies of several dogs for the purpose of determining any possible local cardiac effects have shown only chronic adhesive pericarditis at the site of cardiac incision, and complete occlusion of the azygos vein without dilatation. The endothelial surface of the auricle at the site of incision showed no thrombus formation. The intimal linings of the superior and inferior vena cava at the sites of occlusion were without evidence of damage. In none of the lungs or coronary arteries was there evidence of air embolism.

DISCUSSION

The use of the caval occlusion method has been an aid to the authors in other lines of experimental cardiac surgery. The blind production of interatrial and interventricular septum defects is frequently unsatisfactory and unsuccessful. When such defects are produced in the "dry field," a much better result is accomplished.

Use of the "dry field" allowed the authors to suture atrial septal defects under direct vision. In this maneuver the blood from the left side of the heart was occluded by external finger pressure on the posterior wall of the left auricle.

The action of the tricuspid and pulmonary valves has been observed under direct vision after opening the right ventricle.

CONCLUSION

In this series of experiments it has been demonstrated that in the dog the right heart can be opened, examined and intracardiac manipulation carried out in a "dry field." These procedures are done safely by clamping the superior and inferior vena cavae for periods up to one and one-half minutes, releasing the occlusion of the vessels, and waiting until the heart resumes normal rhythm and rate. The process is then repeated. This has been done 10 consecutive times on each of 10 dogs. The azygos vein was permanently ligated in all dogs. None of the dogs died or developed sequelae. It is felt that this maneuver may be of value in cardiac surgery on human beings.

BIBLIOGRAPHY

1. Dogliotti, A. M., and Ciocatto, E.: The possibility of surgery on the bloodless heart excluded from the circulation in animals treated with ganglionic agents and hypotension, Bull. Soc. Piemont. Chie. 22:649, 1952.
2. Watkins, Elton Jr.; Pomeranz, Alfred A.; Goldsmith, Edward I., and Gross, Robert: Experimental closure of atrial septal defects: technique of an "atrial well" operation, Surgical Forum, Clinical Congress of Amer. Coll. Surg., pg. 247, 1952.
3. Bailey, C. P., et al.: Congenital interatrial communications, Annals of Internal Medicine, 37:888, 1952.
4. Andreasen, A. T., and Watson, F.: Experimental cardiovascular surgery, Brit J. Surg. 39:548-551, 1952.
5. Cohen, M.; Hammerstrom, R. N.; Spellman, M. W.; Varco, R., and Lillehei, C. W.: The tolerance of the canine heart to temporary complete vena cavae occlusion, Surgical Forum, Clinical Congress of Amer. Coll. Surg., pg. 172, 1952.
6. Biglow, W. G.; Callaghan, J. C., and Hopps, J. A.: General hypothermia for experimental intracardiac surgery, Ann. Surg. 132:531, 1950.

Twenty thousand years ago the family was the social unit. Now the social unit has become the world, in which it may truthfully be said that each person's welfare affects that of every other.

—Arthur H. Compton

The Low Salt Syndrome in Children during Hot Weather

Mary E. Evans, M.D.

Kansas City, Kansas

The occurrence of the low salt syndrome as a result of excess sweating and low intake of sodium chloride in hot summer months is not uncommon in children. Very little recognition has been given to this syndrome in children in the literature and none at all in the pediatric textbooks. The clinical diagnosis is often difficult and sometimes missed—with disastrous results. It is important that physicians be familiar with this syndrome, since treatment is highly effective if instituted properly. It is on this account that the following three cases admitted to the K.U. Medical Center this past summer are being reported.

Case 1, D. J., a 14-month-old male, was admitted to KUMC on June 16, 1953, on the neurosurgical service with the chief complaints of drowsiness and lethargy. Two days prior to admission, the child was thrown against the dashboard of the family car; there was no loss of consciousness, but within two hours the child became unusually sleepy. For the 24 hours immediately prior to hospitalization, the child never roused fully, though there was tossing about and willingness to drink water. In this period he was admitted to a local hospital, where he was noted to be anuric and where he was given parenteral fluids. He was transferred to KUMC with the presumed diagnosis of head injury.

His past history was in no way unusual. Physical examination on admission disclosed a well nourished and developed child who appeared adequately hydrated. He tossed about when stimulated, but apparently was unconscious. Positive findings included a bruise on the forehead, large pupils which responded only negligibly to light, equivocal nuchal rigidity, a liver palpable 2½ cm. below the right costal margin, and bilateral Babinski reflexes. The child was afebrile; the pulse rate was 96, and respirations were 18 per minute. Hemoglobin on admission was 12.6 gms. per cent; the total white blood cell count was 5600, of which 60 per cent were polymorphonuclear leucocytes.

This child was treated as a surgical emergency. Prior to surgery, a cutdown on an ankle vein was done; during this procedure the child did become conscious and talked coherently. At surgery, bilateral temporal burr holes were made; no hemorrhage was encountered. Following the operation, the child did not regain consciousness.

The next morning tightness and isolated twitching of the extremities were noted, followed by a convulsion lasting 3 to 4 minutes. He was put in an oxygen tent. Chloral hydrate (grains five) was given rectally. No further convulsions occurred, but the extremities remained tight. The patient then vomited coffee ground material, which was positive to benzedine. Wangensteen suction was started.

Blood sugar was 99 mgm. per cent; CO_2 content of the blood was 25.5 mEq./L. Lumbar puncture revealed 8 white blood cells per cubic mm. in the spinal fluid. The sugar concentration in the spinal fluid was 111 mgm. per cent. The concentration of Cl in the spinal fluid was 450 mgm. per cent. This was the first indication of the real diagnosis of the child's illness.

From the Department of Pediatrics, University of Kansas School of Medicine.

TABLE I

Date	*Solution*	*Route of Adm.*	*Total Vol.*	*Total Salt*
6/16	5% glucose in water	i.v.	200 cc.	0
6/17	5% glucose in water	i.v.	500 cc.	0
	5% glucose in ½ normal saline with 8 mEq. KCl	i.v.	500 cc.	2.2
	plasma	i.v.	250 cc.	1.8
6/18	2½% saline	i.v.	100 cc.	2.5
	whole blood	i.v.	250 cc.	1.8
	2½% saline	i.v.	200 cc.	5.0
6/19	5% glucose in water with 8 mEq. KCl	i.v.	500 cc.	0
	2½% saline	i.v.	200 cc.	5.0
	5% glucose in water with 8 mEq. KCl	i.v.	500 cc.	0
TOTAL			3200 cc.	18.3 gms.

WEATHER BUREAU DATA

Day	*Maximum Temperature*	*Minimum Temperature*	*Max. Humidity*	*Min. Humidity*
6/14/53	94°	74°	88%	41%
6/15/53	102°	74°	85%	33%
6/16/53	88°	69°	82%	42%

(Weather Bureau readings were made at Municipal Airport, Kansas City, Missouri).

Because of continued stiffness of the extremities, avertin per rectum (60 mg., later 40 mg. per kilogram) was given at 4- to 8-hour intervals through the second hospital evening and night. On June 18 the concentration of Na and Cl in the blood was 117 and 76 mEq./L., respectively. Because of the low chloride, a total of 18.3 gms. of NaCl in various solutions was given from June 17 to June 19, with distribution of fluids as indicated in Table I. Within 24 hours of the institution of massive salt therapy, this child was up and about, alert, taking food well, and free from abnormal neurological signs. There was nothing in the child's history that would point toward fibrocystic disease. Recent correspondence with his mother indicates that he is growing well, is active and vigorous, and is without pulmonary or gastrointestinal tract signs.

Case 2, D. D., a 3½-year-old male, was admitted on June 30, 1953, with the chief complaints of anorexia for two days and vomiting for one day. He had fibrocystic disease of the pancreas which had been managed with the aid of terramycin, aerosols, fat-poor diet, vitamins, pancreatin, and residence in Arizona in winter and Canada in summer. At the time of this child's present illness, the family was en route by automobile from Arizona to Canada. Family history was significant in that one sibling died with fibrocystic disease at the age of three years.

Physical examination revealed a thin, chronically ill child, not comatose, with ashen color and cold extremities. Temperature was 99° F., respirations were 30 per minute, and the pulse rate was 120 per minute. Heart tones were poor. Breath sounds were distant over the entire chest. Ribs 10, 11, and 12 were absent on the right, the testes were not palpable, and there were bilateral inguinal herniae. Much fecal material was palpable in the abdomen. Admission laboratory work showed the hemoglobin to be 16.1 gms. per cent; white blood cell count was 17,400, of which 82 per cent were polymorphonuclear leucocytes; Na of the blood was 33 mEq./L.; K of the blood was 3.8 mEq./L.; blood CO_2 combining power was 35 mEq./L.; blood Cl was 62 mEq./L.

Intravenous fluids were begun immediately; a total of 1600 cc. was given in 10 hours. Distribution of fluids is indicated in Table II. Oxygen was administered intermittently by mask and tent. Within eight hours after admission, the patient was taking oral fluids without difficulty, and the day following admission the boy was up, active, and taking regular diet.

TABLE II

Date	*Solution*	*Route of Adm.*	*Total Vol.*	*Total Salt*
6/30	5% glucose in water	i.v.	100 cc.	0
	plasma	i.v.	150 cc.	1.05
	whole blood	i.v.	150 cc.	1.05
	5% glucose in normal saline with 10 mEq. KCl	i.v.	500 cc.	4.5
	5% glucose in water with 10 mEq. KCl	i.v.	450 cc.	0
	5% glucose in normal saline	i.v.	250 cc.	2.25
TOTAL			1600 cc.	8.85 gms.

Case 3, R. G., a 14-month-old female, was admitted to KUMC on August 2, 1953, with the chief complaint of "being cold and blue." This child was said to have been in good health until the day prior to admission, when she became clammy, anoretic and thirsty. On the morning of admission she was taken to a physician who is said to have told the parents that the child had heat stroke, but prescribed no

WEATHER BUREAU DATA

Day	*Maximum Temperature*	*Minimum Temperature*	*Max. Humidity*	*Min. Humidity*
6/28/53	83°	71°	92%	60%
6/29/53	95°	74°	94%	40%
6/30/53	92°	77°	77%	40%

(Weather Bureau readings were made at Municipal Airport, Kansas City, Missouri).

medication. Later in the day she was taken riding by her parents and to a park to play. At this time she was noted to be lethargic. In mid-afternoon she began vomiting phlegm and lost consciousness for a few minutes. She was taken immediately to a local hospital where a complete blood count was done, revealing a high white blood cell count. The parents were told the child might have leukemia, and immediate admission to KUMC was arranged.

On admission no history of recent exposure to infectious diseases or intoxicants could be elicited. There was a history of an illness diagnosed as "asthma," present from birth to 10 months, with no subsequent difficulty. The mother reported that the child had many loose stools if she took freely of usual table foods.

On examination the child was well nourished, well developed, listless, and acutely ill. Her skin was cold and clammy. Peripheral pulses were absent; the anterior fontanelle was depressed; the neck was supple. There was no adenopathy. The chest was clear to percussion and auscultation. The heart rate was regular; heart tones were poor; there were no murmurs. The abdomen was soft, non-tender, and no masses were palpable. The deep reflexes were equal and hypoactive. The child's pulse was 140 per minute, blood pressure was 40/0, respirations were 45 per minute, and temperature was 99.8° F. Admission laboratory work disclosed a hemoglobin of 14 gms. per cent, a white blood cell count of 24,800, of which 72 per cent were polymorphonuclear leucocytes; the CO_2 content of blood was 13.5 mEq./L.

Intravenous fluids were started immediately with distribution as indicated in Table III. Penicillin and streptomycin were started intramuscularly. During the first six hospital hours, the child could be roused and drank freely of a mixture of Darrow's solution and glucose in water. About six hours after admission the child convulsed briefly. Calcium gluconate intravenously was given. The child was placed in an oxygen tent. The patient had been anuric since admission; she was catheterized, but no urine was obtained. Following the convulsion, the child's temperature rose to 103.2° F. and remained at high levels in spite of antipyretic measures for the remaining hours of her life.

In her final 12 hours, the child became opisthotonic with marked rigidity of extremities. Intramuscular phenobarbital was ineffective in relieving the rigidity; rectal avertin was used with satisfactory relaxation of the patient. Terminally there was considerable difficulty with the airway. The child vomited coffee ground material, and frequent suctioning was necessary. Eighteen hours after admission the child expired. She had been anuric from admission even though 1500 cc. of fluids had been given orally and intravenously. It was the clinical impression that she had developed lower nephron nephrosis. It was for this reason that salt-containing fluids were not given in greater amounts. Blood drawn three hours before death revealed NPN of 113 mgm. per cent; Na, 119 mEq./L.; Cl, 66 mEq./L.; K, 6.8 mEq./L. Blood culture was negative at 10 days.

TABLE III

Date	*Solution*	*Route of Adm.*	*Total Vol.*	*Total Salt*
8/2/53	2:1 5% glucose in water Darrow's solution	oral	500 cc.	1.19
	plasma	i.v.	50 cc.	.04
	whole blood	i.v.	200 cc.	1.4
8/3/53	5% glucose in water	i.v.	250 cc.	0
	5% glucose in normal saline	i.v.	250 cc.	2.25
	plasma	i.v.	250 cc.	1.75
TOTAL			1500 cc.	6.63 gms.

Autopsy revealed fibrocystic disease of the pancreas; there was no evidence of lower nephron nephrosis. In the absence of demonstrable kidney lesion, the anuria must be assumed to have been the result of markedly lowered blood volume.

DISCUSSION

The low salt syndrome, as seen in the cases presented here, is the direct result of losses of water and sodium chloride by sweating and of inadequate replacement of these salt losses. Parents are not alert

WEATHER BUREAU DATA

Day	*Maximum Temperature*	*Minimum Temperature*	*Max. Humidity*	*Min. Humidity*
7/31/53	100°	80°	68%	30%
8/1/53	100°	81°	72%	31%
8/2/53	100°	79°	72%	29%

(Weather Bureau readings were made at Municipal Airport, Kansas City, Missouri).

to the degree of their children's sweating, and the need for extra sodium chloride during hot weather is not universally recognized. In hot weather children are driven by thirst to replace their water losses, but, unless there is attention to their need for salt, the fluids used for replacement are likely to be electrolyte-poor. This loss is enhanced by the child's frequent refusal of solids during periods of heat.

That sweat does contain electrolytes sufficient to deplete the extra-cellular fluid if not replaced has been shown by numerous studies. One of the more recent of these was reported by Ladell in 1949;[1] he exercised 31 young, healthy adults in high external temperatures, while keeping careful salt and water balances. As would be expected, he found marked differences among his subjects in their tolerance for the testing situation. Reactions anywhere from "no change" to "fatigue," "edema," "thirst," "marked cramps," and "grey vision" were reported. Subjective symptoms were never observed when blood chlorides were normal. Intracellular overhydration, brought about by ingestion of salt-free fluids during the testing period, markedly increased the subjective symptoms. This obviously is comparable to the child's drinking large quantities of water without adequate salt intake.

In very young children, losses of electrolytes in sweat have been quantitatively determined by Cooke, Pratt and Darrow.[2] These investigators studied four infants kept on metabolic mattresses with analyses of their urine, stools, regurgitated material, and body washings for sodium, potassium, and chloride. This study was carried out with environmental temperatures at 81 and 91° F. Infants' feedings were varied in their electrolyte contents. Hyperpyrexia was not observed at these temperatures. As might be expected, however, on increasing the environmental temperature to 91°, there was immediate increase in the evaporative water loss and in electrolyte loss by way of the skin with the onset of active sweating.

Di Sant'Agnese, Darling, Perera, and Shea reported a striking finding recently.[3] Two groups of individuals, one known to have fibrocystic disease of the pancreas and the other group ill with various other disorders, were subjected for 1 to 2 hours to room temperature of 32.2° C. with 50 per cent humidity. Sweat was collected from the abdominal skin in all cases. The mean volume of sweat was not significantly different in the two groups; markedly different, however, was the chloride content of the sweat. The control group's sweat contained 4-80 mEq./L. of chloride, while that of the group with fibrocystic disease of the pancreas contained 60-160 mEq./L. of chloride. This finding suggests that the results of heat stress in children with fibrocystic disease of the pancreas are much more devastating than in children not ill with this disease.

This is indeed the case. Kessler and Anderson[4] reported 12 cases of heat prostration in New York City during periods of heat in which temperatures ranged from 99 to 107° F. Seven of this group had fibrocystic disease; of the remaining four, three were convalescing satisfactorily from surgery of the gastrointestinal tract, one had cerebral hypoplasia, and one had a congenital cardiac anomaly with cyanosis. Three expired shortly after their admission to the hospital; nine recovered after administration of salt-containing parenteral fluids and antipyretic measures.

The serum chlorides reported in this group ranged from 59 to 81 mEq./L. It is of interest to note that in several instances white blood cell counts on the order of 12,000 to 15,000 were reported, in the absence of any signs of infection. In Case 3 of our series, an elevated white blood cell count was present and had been interpreted at another hospital as a sign of possible leukemia. We would speculate that the leukocytosis may reflect merely adrenocortical response to stress.

In our present series, one child was known to have fibrocystic disease of the pancreas prior to his illness with the low salt syndrome, one child had unsuspected fibrocystic disease of the pancreas found on autopsy, and one child apparently did not have this illness. Certainly every child who tolerates heat poorly should be studied carefully with the possibility of fibrocystic disease in mind, and all infants known to have fibrocystic disease of the pancreas should receive extra sodium chloride.

At the time we saw Case 1, we were unfamiliar with all the aspects of the low salt syndrome. The finding of the low blood chloride was unexpected. We were aware that children in hot weather sweat, are anoretic, have low grade fevers (as reported by Cardullo, 1949),[5] vomit, and are sometimes lethargic. The findings of extreme lethargy deepening into coma, with abnormal neurological signs, opisthotonos, and convulsions are less frequently seen. The low salt syndrome must be included in the differential diagnosis of a child who is lethargic, stuporous, or comatose during periods of hot weather. The determination of the blood chloride is relatively simple, and the correction of this condition can be carried out quickly and easily, unless, as pointed out by Halley,[6] irreversible damage, particularly to the kidney, has already occurred.

As in any other illness, prophylaxis is the best therapy. Friedfeld,[7] among aged patients, helps individuals meet heat stresses by dispensing salt-rich solutions, and daily "heat rounds" in which amounts of sweating and slight temperature variations are noted, with prompt removal of individuals suspected of early heat reactions to cooler environmental temperatures. Cardullo[5] reports similar measures in hos-

pitalized infants in salting formulae and leaving infants unclothed.

It has become our custom to give hospitalized infants and young children mixtures of Darrow's solution and glucose water, instead of ordinary water, between feedings, and to give older children anywhere between 0.5 gm. and 1.5 gms. per day of salt in addition to their regular diet. In the case of children who are not hospitalized, parents should be instructed to encourage drinking fluids rich in electrolytes (or to administer salt tablets), particularly when children are eating poorly, and to watch carefully for excessive sweating or lethargy.

SUMMARY

Three cases of low salt syndrome have been presented. The etiology and symptomatology, with special reference to the correlation between the syndrome and the presence of fibrocystic disease of the pancreas, have been discussed. It is concluded that the low salt syndrome is not necessarily the inevitable consequence of excessive sweating, but rather, the inevitable consequence of indequate replacement of water and electrolyte losses through sweating. Failure to replace these losses is often due to failure to recognize the need for replacement.

BIBLIOGRAPHY

1. Ladell, W. S. S.: Heat cramps, Lancet 2:836, 1949.
2. Cooke, R. E.; Pratt, E. L., and Darrow, D. C.: Metabolic response of infants to heat stress, Yale J. of Biol. & Med. 22:227, 1950.
3. Di Sant'Agnese, P. A.; Darling, R. C.; Perera, G. A., and Shea, Ethel: Abnormal electrolyte composition of sweat in cystic fibrosis of pancreas, Pediatrics 12:549, 1953.
4. Kessler, W. R., and Anderson, D. H.: Heat prostration in fibrocystic disease of the pancreas and other conditions, Pediatrics 8:648, 1951.
5. Cardullo, H. M.: Sustained summer heat and fever in infants, J. Pediat. 35:24, 1949.
6. Halley, H. L.: The salt depletion syndrome, So. Med. J. 45:153, 1952.
7. Friedfeld, Louis; Prophylaxis and treatment of heat-reaction states, N. E. J. M. 240:1043, 1949.

The Use of Trypsin in Rheumatoid Arthritis: A Clinical Study

William H. Goodson, Jr., M.D., Donald L. Rose, M.D., and William S. Alyea, M.D.

Kansas City, Kansas

Innerfield, Angrist, and Schwarz[1] have reported marked clinical improvement in patients with rheumatoid arthritis following the intravenous administration of trypsin. When it was indicated[2] that the intramuscular route was probably as effective as the intravenous, a group of six patients with rheumatoid arthritis volunteered to submit to intramuscular administrations of trypsin in oil.

All patients presented evidence of rheumatoid arthritis according to the standards of the New York Rheumatism Association.[3] All presented moderate activity of the disease with no more than moderate x-ray evidence of joint damage, so that hope for definitive improvement was a legitimate possibility. All had been under observation by the authors for at least six months and had been stabilized during this period on a consistent medical program of analgesics, desoxycholic acid, vitamin D, other vitamins, and simple physical therapy. The medical program remained unaltered during the period of the study, with the exception of one case, other than by the administration of trypsin in oil injections. It was felt that the effects of the trypsin could best be evaluated if all other variables were as constant as possible.

The method of administration of the trypsin was the same in each instance: 2 mgms. were given intragluteally morning and evening the first day; 3 mgms. were given intramuscularly twice daily for the next five days. After the sixth day, the patients were reevaluated. Treatment was then resumed in larger dosage on alternate days for the following three weeks. The first two doses at the 48-hour interval were 10 mgms. each; since this increased dosage produced severe local pain without evidence of commensurate improvement in the arthritic symptoms, the individual dosage was reduced to 5 mgms. for the remaining two and one-half weeks of treatment. Complete blood counts were made, including differentials and platelets, and other laboratory studies were erythrocyte sedimentation rate, cephalin flocculation test, and complete urinalysis. These laboratory determinations were made just prior to, at

Dr. Goodson is from the Department of Medicine, University of Kansas School of Medicine. Dr. Rose and Dr. Alyea are from the Department of Physical Medicine.

The trypsin in oil was obtained from the National Drug Company (Parenzyme in oil, 2 mgms. trypsin in 1 cc. of sesame oil).

the end of the first week of, and at the conclusion of the four-week treatment period.

RESULTS

Each of the patients commented on an increased sense of well being during the first week of treatment. At the conclusion of the treatment period, only one patient felt that this had continued. Similarly, there were minor improvements in joint mobility and reductions in pain during the first week of treatment, but the objective improvement at the end of the period was negligible except in this single case. This exceptional patient enjoyed mild progressive improvement in pain, strength, and mobility which has continued during a six-month post-treatment period; in contrast to the other patients, he received phenylbutazone, 100 mgms. twice daily, as a part of his medical program.

One patient misunderstood her instruction, as was learned subsequently, and discontinued all other treatment during the experimental period. Even while receiving trypsin on alternate days, she developed acute and virtually incapacitating recrudescence of heat, pain, swelling, and stiffness in her knees and wrists. These symptoms promptly subsided when her previous treatment was resumed.

In three patients there was no significant change in the red blood cell count. In two patients there was a fall in hemoglobin value of 6 to 8 per cent. The sixth patient developed a transient painless hematuria four days after the conclusion of the study, and the hemoglobin value fell from 64 per cent to 38 per cent; no explanation for this phenomenon was discovered, although he was hospitalized and examined carefully from the urologic standpoint. There were no consistent alterations in the blood sedimentation rates, which were all high. The white blood cell count in one patient fell from 4,000 per cubic millimeter before treatment to 1,600 at the end of the first week, but rose to 3,400 as treatment was continued. The cephalin flocculation test ranged from 2+ to 4+ at the beginning of treatment in three patients; at the end of one week in the same patients a normal 2+ and 1+ value was reported.

CONCLUSIONS

1. Six patients with typical rheumatoid arthritis were given trypsin in oil intramuscularly over a four-week period.

2. In five of the six patients, aside from a temporarily increased sense of well being during the first week of treatment, no subjective or objective signs of improvement were noted. The sixth patient enjoyed steady improvement during and subsequent to the treatment period; phenylbutazone was included in his medical program.

3. The development of transitory leucopenia in one case and severe anemia with hematuria in another, shortly after treatment was discontinued, suggest that the administration of trypsin should be carried out only when careful supervision is possible.

4. Trypsin in oil administered intramuscularly under the circumstances outlined does not appear to be useful in the treatment of rheumatoid arthritis.

BIBLIOGRAPHY

1. Innerfield, I.; Angrist, A., and Schwarz, A.: Parenteral administration of trypsin, J.A.M.A. 152:597, 1953.
2. Innerfield, I.: Personal communication.
3. Primer on the Rheumatic Diseases, J.A.M.A. 152: 323, 405, 522, 1953.

IN FUTURE ISSUES OF THE JOURNAL

Two scientific papers scheduled for publication in this issue of the JOURNAL were omitted because of lack of space. They will be published later. The papers are:

Complete Heart Block With Morgagni-Adams-Stokes Syncope Treated with Large Doses of Epinephrine: A Case Report—William E. Larsen, M.D., Departments of Medicine, Veterans Administration Hospital, Kansas City, Missouri, and University of Kansas Medical Center, Kansas City, Kansas.

Report on the Tubeless Gastric Analysis—R. L. Van Citters, M.D., and Mahlon Delp, M.D., Department of Medicine, University of Kansas School of Medicine.

A Twenty-Four-Year Review of Cesarean Sections at Kansas University Hospitals

J. C. Schroll, M.D., and L. A. Calkins, M.D.

Kansas City, Kansas

The rising incidence of cesarean sections in most areas of our country is well recognized. In view of this, it was thought there might be some value in evaluating our own cases. In the United States today, 1.5 to 2 per cent of all pregnant women will be delivered by cesarean section.[1, 2] Every year the obstetrical journals publish papers comparing present statistics with previous results. Many articles justify the increase in incidence of cesarean sections on the basis of broadening the indications for this type of delivery.[3, 6] This broadening of indications, however, has been accompanied by the elimination of a few indications that are no longer tenable with our present knowledge of medical practice.

There are a number of large series of cesarean sections in recent literature which report little or no mortality and a significant increase in fetal salvage. This can be attributed to increasing use of antibiotics, the availability of blood banks, better anesthesia, and many other factors, all of which have resulted in a liberalization of the indications for cesarean section.

D'Esopo[3] has shown that abdominal section is slowly replacing the formidable midforceps delivery, and he feels an incidence rate of approximately 6 per cent cesarean sections should be adequate to eliminate the trauma, both maternal and fetal, caused by difficult vaginal operations. There seems to be general agreement that increasing use of abdominal delivery in many obstetrical problems is indicated, but that over-utilization of the operation is still a problem. Maternal mortality for abdominal delivery continues to carry 10 times the mortality of vaginal delivery.

Material: This review includes 471 consecutive cesarean sections among 15,871 deliveries done between September 1929 and September 1953. All operations were performed either by a member of the obstetric staff or by obstetric residents under supervision. The majority of patients received adequate prenatal care, although there were many exceptions among clinical or ward-type patients. The ward service is considerably larger than the private services.

Incidence: Figure 1 shows the incidence for five-year periods, which affords a better indication of the true incidence due to the larger number of considered cases. One can readily note that there has been a significantly increasing trend only in the post-war period. The last period, although short of five years, would indicate our approximate present rate of cesarean sections to be 4 per cent. It would appear that the greatest increase in incidence has occurred in the last time period, which is only 44 months and shows a 1 per cent increase over the previous five-year period and approximately 1.5 per cent increase over the 20 years preceding 1950. The incidence rate for the entire series was 2.9 per cent. We feel the trend to an increased incidence seen here is entirely justifiable and perhaps has played some role in the increased fetal salvage at this institution.

FIGURE 1

INCIDENCE OF CESAREAN SECTIONS FOR FIVE-YEAR PERIODS

	Deliveries	*C.S.*	*Per Cent*
1930-1934*	1595	27	1.7
1935-1939	2006	54	2.2
1940-1944	2777	67	2.4
1945-1949	4454	127	2.9
1950-1953**	5039	196	3.9
TOTAL	15,871	471	2.9

* Includes the last four months in 1929
** Includes the first eight months only of 1953

Parity: The parity of these patients is recorded in Figure 2. Approximately one-third of the patients were in their first pregnancy, and nearly two-fifths were having their first delivery. There was one patient who had had 13 previous children.

FIGURE 2

	Parity	
Total	471	100%
Primipara	151	32.1%
Multipara	320	67.9%

PREVIOUS DELIVERIES

Number of Deliveries	*Number of Patients*	*Per Cent*
None	181	38.4
One	139	29.5
Two	70	14.9
Three	33	7.0
Four	19	4.0
Five	8	1.7
Six	4	0.8
Seven	7	1.5
Eight to Thirteen	12	2.5

Labor and Membranes: Data concerning labor and

Department of Obstetrics, University of Kansas School of Medicine.

membranes is presented in Figure 3. Labor was present in some degree in 16.4 per cent of the patients. Up to recent years, approximately 95 per cent of our abdominal deliveries were elective. These figures reflect an increasing use of trial of labor in many patients these last several years, patients who would have previously had elective sections.

FIGURE 3
LABOR AND MEMBRANES

	Number	*Per Cent*
Labor:		
No labor	394	83.6
Less than 6 hours	21	4.5
6 to 11 hours	10	2.1
12 to 17 hours	13	2.7
18 to 23 hours	7	1.5
24 to 29 hours	5	1.0
30 to 36 hours	7	1.5
36 to 48 hours	5	1.0
48 hours and over	7	1.5
Membranes:		
Intact	413	87.7
Ruptured:		
5 hours or less	20	4.2
6 to 9 hours	10	2.1
10 to 14 hours	3	0.6
15 to 19 hours	3	0.6
20 to 24 hours	0	0.0
25 to 29 hours	1	0.2
30 to 40 hours	8	1.7
40 hours and over	10	2.1
Unknown	3	0.6

The membranes were ruptured for varying lengths of time before surgery in 12.3 per cent of all cases. There were 10 patients with ruptured membranes for 40 hours or more, and in a few of these it was for several days.

Indications: In tabulating the indications for cesarean section, the primary indications (Figure 5) were

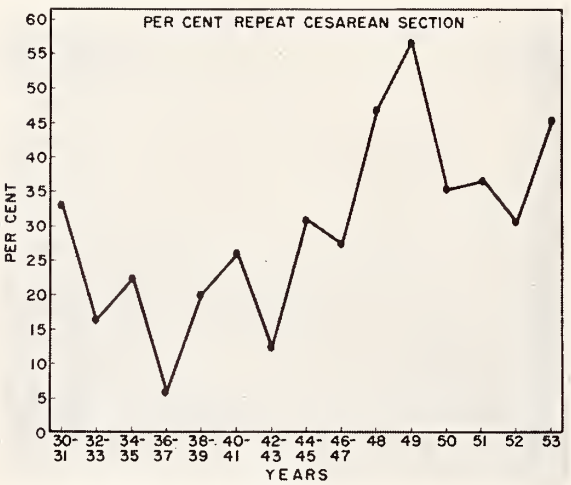


FIGURE 4

separated from those that seemed to be secondary. Those considered as primary were any indications that would necessitate in themselves a cesarean section, and the secondary indications were those that would not, except in combination with other indications, invoke the necessity of a cesarean section. We believe this has made the following tables more meaningful.

Repeat cesarean section was a primary indication in 32.3 per cent of our patients. Figure 4 is a graph illustrating the somewhat erratic but definite upward trend in the number of repeat sections. In 1949 they composed over 50 per cent of the sections done that year. At no time in recent years have they been less than 30 per cent. They now compose nearly 40 per cent of all sections.

How many of these repeat cases could have been considered eligible for a subsequent vaginal delivery? There were 83 of the 152 patients in whom the primary reason for their first cesarean section did not exist at the time of their subsequent deliveries. Of the 471 cesarean sections in this series, 9 known patients have subsequently delivered 11 times vaginally, including one set of twins. This is not a true picture in that many of the recent sections were in patients who have not had adequate time for subsequent pregnancies, and an unknown number of patients have had subsequent deliveries elsewhere. This indicates that in this center we do not adhere closely to the dictum, "Once a cesarean section, always a section." The New York Lying-In Hospital and the Margaret Hague Maternity Hospital[8] have reported that 38.7 and 35.8 per cent, respectively, of their women with previous sections are delivered vaginally. Although we are more willing to give these patients a trial of labor now than previously, we have not reached the point of letting the majority of these patients with previous sections labor as a trial.

FIGURE 5
PRIMARY INDICATIONS FOR CESAREAN SECTION

Indications	*Occurrence*	*Per Cent*
I Repeat cesarean sections	152	32.3
II Cephalopelvic disproportion	156	33.1
III Other pelvic obstructions	44	9.3
IV Toxemias	47	9.9
V Hemorrhage	60	12.7
VI Firm cervix or cervical dystocia	38	8.1
VII Thin uterine scar diagnosed clinically	20	4.2
VIII Myomectomy previously	5	1.0
IX Diseases	64	13.6
X Abnormal presentation	7	1.5
XI Miscellaneous	30	6.4

A slightly larger but approximately equal group in actual numbers to the repeat section group is the cephalopelvic disproportion patients. There were 72 of the 156 patients with small measurements of outlet or midpelvis, 15 with small inlet measurements, and 45 with a generally contracted pelvis. All of these patients, with only rare exceptions, had x-ray pelvimetry. There were an additional 8 patients with pelvic deformities due to fracture, disease, or unknown causes. The monsters, two hydrocephalic, and two of other types were also placed in this disproportion group.

In the third group labeled as "other pelvic obstructions" were 9 patients with obstructive fibroids, 3 with ovarian masses, and 12 with old third degree lacerations of the perineum. There were an additional 20 patients with cervical carcinoma or previous cervical operations.

Severe toxemia was the primary indication for abdominal delivery in nearly 10 per cent of the 471 patients. Three of the 47 patients had eclampsia. Only those patients with toxemia who were not responding to conservative management and in whom induction of labor seemed out of the question received a cesarean section.

Among the hemorrhage patients were 18 with central and 21 with marginal placenta previa. The 20 patients who were delivered by section for premature placental separation represent a rather small minority in comparison with the vaginally delivered patients with this complication.

In this institution we have recognized the so-called "firm cervix" for many years, but only recently have we become fully aware of its significance in regard to fetal mortality. It has caused more fetal deaths than placenta previa in this hospital. The statistics and importance of this usually unrecognized entity will be published later this year by Dr. Calkins.

We now have the opinion that cesarean section is indicated with these conditions: (1) a firm cervix which does not soften as labor progresses; (2) poor pains which show no tendency to improve as labor progresses; (3) persistently high station, and (4) if the first stage will obviously exceed 30 hours.

We feel it is necessary to see our patients early in labor and to follow them closely throughout their labor, which probably explains why we are impressed with this entity.

At surgery, 13 of the 20 patients with a clinical diagnosis of a thin uterine scar showed a very poor and thin scar which would not have tolerated labor. Three patients had intact scars with no defects. The condition of scar in 4 patients is unknown. However, there were 6 patients with poor scars and 4 with incomplete rupture of old scars that were not diagnosed clinically before surgery. It appears that our showing is no better than others in evaluating uterine scars. Most authors frankly state they are as frequently wrong as right in their diagnoses of weak uterine scars.

In the disease indication group, rheumatic heart disease was the primary indication in 26 patients. The majority of these cases occurred before the last 5 to 10 years. Only the most severe rheumatic heart disease patient would be sectioned in this clinic now. There were 13 severe diabetic patients delivered by the abdominal route. A number of these were severe juvenile-type diabetics, and some had poor obstetrical histories. No cesarean section for tuberculosis has been done in recent years.

The abnormal presentation cases were mostly transverse lies. Brow presentation was noted twice and twins once with both breeches lodged in the pelvis. These latter three cases were non-elective cesarean sections.

Those patients in the miscellaneous group were: psychiatric, 1; elderly primigravida, 2; poor obstetrical history, 8; premature rupture of membranes, 3; acute hydramnios, 3; fetal distress, 5; others, 8.

The secondary indications are listed in Figure 6. Those showing the most common occurrence were unfavorable cervix, abnormal presentation, and the miscellaneous group. Mild pre-eclampsia and poor obstetrical history were factors in 10 patients.

FIGURE 6

SECONDARY INDICATIONS FOR CESAREAN SECTION

Indications	*Occurrence*	*Per Cent*
I Cephalopelvic disproportion	6	1.3
II Other pelvic obstructions	1	0.2
III Mild pre-eclampsia	10	2.1
IV Unfavorable (unprepared) cervix	33	7.0
V Diseases	10	2.1
VI Abnormal presentation	16	3.4
VII Miscellaneous	32	6.8

Types of Operations: The types of cesarean section done are shown in Figure 7. It is not clear how some authors have counted their low segment procedure with a vertical incision, so these are listed separately. The high incidence of classical sections is in marked contrast to most reported series. At present we do many more Phaneuf (transverse cervical) sections than classical sections. Those patients having a hysterectomy total 4.4 per cent. The reasons given for those patients having a Porro were fibroids in 6 patients, a Couvelaire uterus that would not respond in 2, an abnormal uterus in 2, and thin old scars in 2. Uterine rupture, frozen pelvis due to acute myelogenous leukemia, Grade I carcinoma of cervix, previous cervical amputation, and one kidney were each listed as the reason in one case. Those patients having a total hysterectomy were listed as carcinoma of cervix Grade I, cervical fibroid, and previous cervical amputations.

FIGURE 7

TYPE OF CESAREAN SECTION

Type	*Patients*	*Per Cent*
Classical	344	73.0
Low segment, midline incision	52	11.0
Low segment, transverse incision (Phaneuf)	54	11.5
Porro	17	3.6
Total hysterectomy (Bishoff)	4	0.8

Anesthesia: The incidence and types of anesthesia are listed in Figure 8. Since no detailed study of

natal deaths was done, no attempt was made to determine the effect of different anesthetic agents and methods upon the mother, fetal resuscitation, or fetal mortality. The great majority of the spinal anesthetics were given in the last 5 or 6 years. Most of the local anesthetic cases were done in the past year. Previous to that time, most sections were done under general anesthesia of different types.

FIGURE 8
TYPE OF ANESTHESIA

Anesthesia	*Patients*	*Per Cent*
Spinal	203	43.1
Spinal with gas	19	4.0
Cyclopropane	111	23.6
Cyclopropane-Nitrous	11	2.3
Nitrous	39	8.3
Local	9	1.9
Other	56	11.9
Unknown	23	4.9

Transfusions: As a matter of interest, the number of transfusions that these patients received during their hospitalization was recorded. Two hundred ninety-two patients, or 62 per cent, received no transfusions. Those receiving only one transfusion constituted 22.7 per cent, and an additional 12.3 per cent received two or three transfusions.

Two patients received quite large amounts of blood. One was a 23-year-old colored primigravida who was admitted at 41 weeks gestation with a diagnosis of placenta previa. She was allowed to labor for one hour and was then sectioned. A uterine pack was used at surgery, but the patient continued to bleed excessively and it was necessary to do a total hysterectomy four hours later because of a hemorrhagic diathesis. Both mother and child lived. This patient received 15 pints of blood. The other patient was also a 23-year-old primigravida with a frozen pelvis from acute myelogenous leukemia. A Porro section was done at 34 weeks gestation because of a rapidly worsening course. She received a total of 17 pints of blood, three of which were given in the postoperative period. She expired on the second postoperative day.

FIGURE 9
POSTOPERATIVE MORBIDITY (100.4 Degrees)

*Days of Fever**	*Patients***	*Per Cent*
None	129	27.7
One	155	33.3
Two	71	15.2
Three or more	111	23.8

* Consecutive days of fever
** 466 patients

Morbidity: Figure 9 shows the incidence of postoperative morbidity. Slightly more than one-quarter of the patients had no morbidity. An additional one-third of the patients had only one day of fever, which in the majority of cases occurred on the first postoperative day. Reviewers of cesarean section cases consider patients having fever only on the first postoperative day as insignificant and do not record them in morbidity statistics. If we did, this 61 per cent of our patients had no morbidity. Another 15 per cent of the patients had only two days of fever. The more severe cases of morbidity total 23.8 per cent. In the last five-year period, only 18 per cent have had a fever of three or more days.

The bar graph of Figure 10 shows the marked change that has come about since the advent of use of antibiotics. We believe that the decreasing postoperative morbidity in the last 12 years can largely be accounted to better pre- and postoperative care

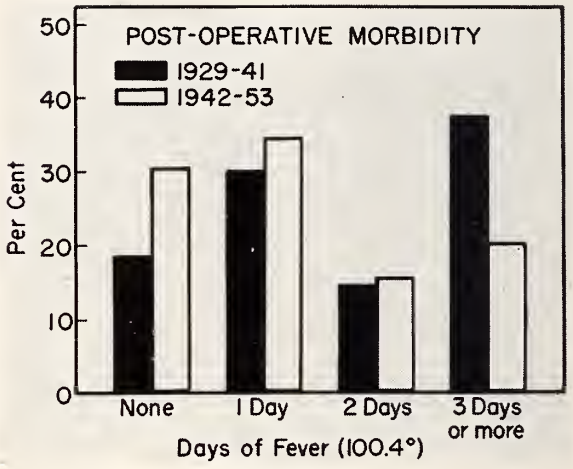


FIGURE 10

with fluid and electrolyte balance and the more prevalent use of transfusions and prophylactic antibiotics. The difference in the more severe morbidity column would probably more directly point to antibiotic usage, although many other factors would also have to be included. In comparing our incidence of morbidity of any two consecutive days after the first postoperative day, which is the way it is usually recorded, with previous reports, we seem to have an unusually high incidence. If only the last five years (which are not shown in figures) are considered with those from other hospitals covering roughly the same period, we have the following: Jones[9] reported an unusually low rate of 15 per cent from the Charlotte Memorial Hospital, Charlotte, N. C. Northwestern University Hospitals[10] reported 22 per cent. Our incidence is 34.6 per cent. No statistical correlation could be found in this small series between degree of morbidity and the duration of ruptured membranes.

Complications: Postoperative complications are listed in Figure 11. Stastically, only two are of real importance. The first is all cases of morbidity in

which no clinical diagnoses could be made. The second is urinary tract infections. It is gratifying to note that in the past five years the percentage of patients with no postoperative complications is approaching twice the figure that our series showed in the first 12 years. It also appears that we are improving our ability to diagnose the cause of morbidity more prominently in the last 12 years than in the first 12 years of this review.

FIGURE 11
POSTOPERATIVE COMPLICATIONS

Complication	*1929*-1941*	*1942-1947*	*1948-1953*
None	16.3%	23.8%	27.2%
Morbidity, cause unknown	43.3	23.8	23.0
Urinary tract infection	8.7	29.5	31.9
Others	31.7	22.9	18.0

Others Include:	*Patients*	*Per Cent*
Wound infection	9	1.9
Wound dehiscence	5	1.1
Pneumonia	17	3.6
Upper respiratory infection	8	1.7
Paralytic ileus	13	2.8
Endometritis	8	1.7
Atelectasis	9	1.9
Others	56	11.9

* From September 1, 1929 to January 1, 1941.

Fetal Mortality: The graph in Figure 12 shows the cesarean section and fetal mortality incidence with gestation. The greatest percentage-wise loss was in the most premature babies, as expected. The critical weight for the premature infants in this series was somewhere between 1750 and 2000 grams. The 3000 to 3250 gram group showed the best survival rate in this series. There appears to be an increase in fetal loss in larger babies, but the small numbers involved reduce the significance of this observation. Fetal mortality is practically zero at 38 weeks, after which time it again shows a progressive rise up to 41 weeks gestation. The large majority of these mothers had

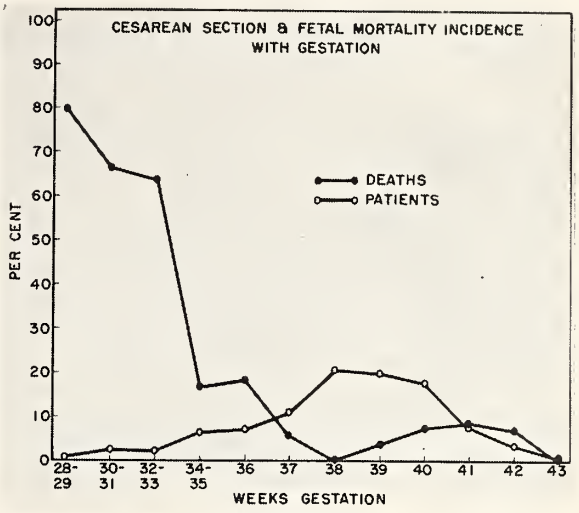


FIGURE 12

their abdominal deliveries between the 37th and 40th weeks.

It would appear from this graph that the babies in our series had their best chance for survival at the 38th week. This statement has been made many times previously in the literature. For diabetic mothers an even earlier date seems most advisable. Omitting the congenital anomalies incompatible with life, our current rate of fetal loss is 7.2 per cent.

Maternal Mortality: The corrected maternal mortal-

FIGURE 13
MATERNAL MORTALITY CASES

Case	*Year*	*Age*	*Parity*	*Indication*	*Cause of Death*
E.B.**	1931	24	0	Rheumatic heart disease with severe cardiac decompensation	Cardiac failure at onset of surgery
G.B.	1937	23	3	Fracture C_6 with only diaphragmatic breathing	Bronchiectasis, pneumonia, pulmonary edema, nephritis
E.L.**	1938	21	0	Severe pre-eclampsia with possible abruptio placenta	Acute peritonitis, hepatitis, toxic nephrosis
J.J.	1944	32	4	Comatose from subarachnoid hemorrhage	Thrombosis of choroid plexus with bilateral intraventricular hemorrhage
B.B.**	1947	34	0	Prolonged labor without progress with firm cervix	Wound dehiscence 9th p.o. day. Died 13th p.o. day from perforation of cecum, acute peritonitis, hepatitis
M.J.**	1948	19	0	Fulminating pre-eclampsia	Eclampsia, pulmonary edema
E.J.	1950	23	0	Frozen pelvis from tumor	Acute myelogenous leukemia on 2nd p.o. day
R.U.	1950	29	0	Onset of labor following craniotomy for malignancy	Meningioma, died 9th p.o. day
M.M.	1951	23	2	Moribund patient	Acute bulbar poliomyelitis
V.S.**	1951	33	1	Prolonged labor with firm cervix & borderline pelvis	Respiratory paralysis from spinal anesthetic

** Considered avoidable deaths

ity incidence of this series of 471 patients is 1.06 per cent. The mortality cases are shown in Figure 13. The first patient (E. B.) was a grade IV cardiac patient in pulmonary edema with a blood pressure of 198/35. Section was done to save the baby, which was, however, a stillborn. Fetal heart tones were last heard two hours prior to surgery, but the mother's respirations obliterated the heart tones after this. This should probably be classified as an unavoidable death.

Cesarean section was done on the second patient for the baby which survived. The mother's death was imminent.

The third patient (E. L.) had a hard tetanic uterus, and abdominal delivery was decided upon for the possible abruptio placenta which was not found. The infant was a hydrocephalic and died the first day.

The fourth patient had a postmortem cesarean section, and a good baby that survived was obtained.

Good babies were obtained with the next four patients. The neurosurgical consultants insisted upon delivery by cesarean section in the eighth patient (R. U.).

The ninth patient (M. M.) had a temperature of 110 degrees, and the infant was nearly too hot to hold in the operator's hands. If surgery had been done four hours earlier, the infant might have lived.

The last patient's death (and fetal loss) was directly due to the anesthetist's carelessness. Cardiac massage was done immediately, but patient never responded.

SUMMARY

1. A 24-year review of 471 consecutive cesarean sections with an incidence rate of 2.9 per cent is presented.

2. In a clinic with a considerable amount of referred pathology, an incidence of four to five per cent is probably justified.

3. It is no longer necessary to do a high percentage of elective sections. Trial of labor can be utilized much more freely.

4. Repeat cesarean section need not be routine, particularly if the operating room and operating team are immediately available.

5. Low cervical (longitudinal incision) is often not feasible as an elective procedure.

6. Increasing use of local anesthesia is advisable. The anesthetist is, however, more important than the agent.

7. Abdominal delivery is still 10 times as dangerous (maternal mortality) as vaginal delivery.

BIBLIOGRAPHY

1. Wilson, A. L.: Am. J. Obst. & Gynec. 62:1225, 1951.
2. Eastman, N. J.: William's obstetrics, ed. 10, New York, Appleton-Century-Crofts, Inc., p. 1101, 1950.
3. D'Esopo, D. A.: Am. J. Obst. & Gynec. 59:77, 1950.
4. Dyer, I., and Nix, F. G.: New Orleans M. & S. J. 104:266, 1952.
5. Kistner, R. W.: Am. J. Obst. & Gynec. 61:109, 1951.
6. Zarow, G. S.: Am. J. Obst. & Gynec. 63:122, 1952.
7. Douglas, R. Gordon: Tr. Fifth Am. Congress on Obst. & Gynec. (supp. vol. Am. J. Obst. & Gynec.) 64A:150, 1952.
8. Cosgrove, Robert A.: Tr. Fifth Am. Congress on Obst. & Gynec. (supp. vol. Am. J. Obst. & Gynec.) 64A:545, 1952.
9. Jones, O. Hunter: Am. J Obst. & Gynec. 66:747, 1953.
10. Zettelman, H. J., and Bowers, V. M.: Am. J. Obst. & Gynec. 65:953, 1953.

All the religious leaders, however far apart in time and place, and however impossible it was for them to communicate and compare their thoughts, have arrived at the conviction that there is a divine purpose in life. This conviction springs as naturally from a devout mind as an oak tree springs from an acorn. It is the result of the spontaneous flowering of human thought. The seed has an instinct to grow into a plant. And man has an instinct to blossom into faith. . . . He is born, as every religious teacher has observed, with an impulse to make himself useful to his fellow men.

—*Henry Thomas*

THE MONTH IN WASHINGTON

Editor's Note. The following summary of Washington news was prepared by the Washington office of the A.M.A. for distribution to state and regional medical journals.

Some parts of the Eisenhower administration's broad health program are making good progress on Capitol Hill, while others are virtually standing still or bogged down in the technical complications that are always a threat to new legislation. Well ahead of the other proposals, and possibly destined for enactment, are bills to broaden the scope of the Hill-Burton hospital construction law and to liberalize income tax deductions for medical expenses.

The House Interstate and Foreign Commerce Committee, under chairmanship of Rep. Charles Wolverton (R., N.J.), wound up its long fact-finding study of voluntary health insurance plans and immediately started hearings on the Hill-Burton changes. The purpose is to amend the Hill-Burton law so that it can be used to disburse federal grants to states for construction of health facilities that do not qualify as "hospitals." The administration is anxious to stimulate the building of more nursing homes, hospitals for the chronically ill, diagnostic or treat ment centers, and rehabilitation facilities.

An initial appropriation of $2 million would be authorized for surveys and planning, and $60 million annually for three years of construction. Per capita income as well as population would be used to determine a state's share, as under the present Hill-Burton program.

At the House hearing, crowded into two days, the construction program was endorsed at least in principle by every witness, except the representative of the American Association of Nursing Homes. Because the program is limited to non-profit sponsors, members of this group could not receive grants. Their spokesman said long-term loans through the Small Business Administration would help solve their problem.

The A.M.A. recommended passage of the bill, but urged that facilities for the chronically ill and the handicapped be "part of or near a conventional hospital," and that facilities of all types be open to the entire community without discrimination, as in the present Hill-Burton law. (It is likely hearings also will be held on this legislation in the Senate.)

The House Ways and Means Committee, meanwhile, was giving its approval to a new income tax provision that would allow the deduction of medical expenses if they exceed three per cent of adjusted gross income, rather than five per cent under present law. The present maximum limitation would be doubled, and the deduction of travel expenses allowed where travel is prescribed by a physician. These changes—a long-time A.M.A. goal—are embodied in the omnibus tax readjustment bill.

President Eisenhower's proposal for federal reinsurance of voluntary health plans has not been able to follow the steady course on which it first appeared to be embarked. At the House hearings, none of the spokesmen for the large organizations in the health fields—A.M.A., Blue Cross and Shield, American Hospital Association—was willing to endorse the plan. Like the A.M.A. spokesmen, most of them wanted first to examine the actual administration bill, which at that time had not been introduced. From the Blue Cross, however, came a suggestion that the idea be tried out experimentally.

Spokesmen for national labor organizations expressed mixed reactions, with some maintaining that reinsurance was a poor substitute for what they believe the country really needs—national compulsory health insurance.

The administration's health budget for the next fiscal year, starting July 1, calls for a slight overall reduction. The regular Hill-Burton program, currently operating on $65 million, would get $50 million (any appropriation to start the proposed expanded construction would be in addition). Relatively sharp reductions would be made in funds for venereal, tuberculosis, and communicable disease control, in line with the policy of shifting this responsibility to the states. The various research institutes would receive about what they are now spending.

One of the few new items is for $7.8 million, estimated as necessary for the extra cost of enlarging the federal program of vocational rehabilitation. Legislation authorizing the expansion is awaiting Congressional action. The administration hopes gradually to increase the number of persons rehabilitated annually from the current 60,000 to 200,000. While the program is being stepped up, one of its goals would be to induce states to increase their spending until eventually their appropriations match the federal. Like most of the President's health program, the rehabilitation effort has the support of the A.M.A.

Conferences between A.M.A. officials and administration leaders are continuing. Latest sessions were with Secretary Hobby, concerning her department's legislative plans; with VA Administrator H. V. Higley, on treatment of non-service connected cases; and with Admiral Arthur W. Radford, chairman of the Joint Chiefs of Staff, and Dr. Frank Berry, assistant defense secretary for health and medical matters.

PRESIDENT'S PAGE

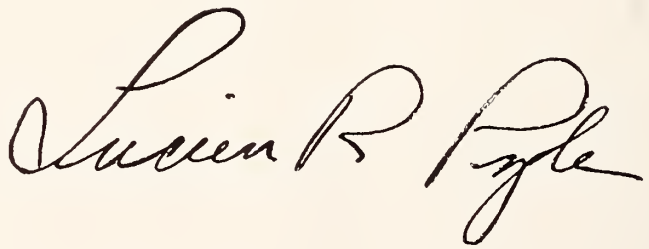
DEAR DOCTOR:

In March of 1947, the first University of Kansas School of Medicine issue of the JOURNAL OF THE KANSAS MEDICAL SOCIETY was published. The fact that each year since that time the Editorial Board and the Medical School have seen fit to continue with that issue speaks for itself.

All matters pertaining to education and advancement in our state should revolve around the hub of our schools and institutions of higher learning. In the case of medicine, that institution is the University of Kansas School of Medicine.

As the science of medicine becomes more detailed and complex, the University of Kansas School of Medicine becomes more essential to each and every one of us. It has met the challenge, not only by offering many courses for continued study at the school, but it has also sent the courses to us right in our own community, in the form of circuit courses. The popularity of this form of continued study in Kansas is attested by the fact that the University of Kansas School of Medicine ranked with the highest in enrollees for courses in postgraduate education last year.

No one can foresee the future, but we can envision the future in the light of past experiences. The sound building and planning of undergraduate and continued medical education, closer and more extensive co-operation of the medical profession with all phases of that educational program, will cause the School of Medicine to grow and prosper. Thus, it will more adequately serve its rightful place in ministering to the health needs of the people of Kansas.



EDITORIAL COMMENT

Eighth Annual K.U. Issue

This, the March 1954 issue of the JOURNAL, is the Eighth Annual University of Kansas School of Medicine Number. Members of the Editorial Board take pleasure in repeating this salute to our state medical school.

Although the JOURNAL seldom goes to press without including some contribution from the school, this issue includes scientific material from that source only. The work of assembling the material was done by Dr. Glen R. Shepherd, an associate editor of the JOURNAL who has ably served in that capacity in former years.

Progress at the Medical School

Editor's Note: The following editorial was prepared for the JOURNAL *by Dr. W. Clarke Wescoe, dean of the University of Kansas School of Medicine.*

One year ago I had the pleasure of writing a progress report on your school of medicine for our JOURNAL. Since that writing many things have happened and additional progress has been made. Therefore, rather than just write a word of welcome in connection with the appearance of this annual medical school issue, I have decided to bring the progress report up to date for you.

Legislative Action. When last I wrote, the legislature was meeting in biennial session and was considering the appropriation requests of the University of Kansas Medical Center. The full membership of the Ways and Means Committees of the House and the Senate visited with us and toured the center. The appropriations, as they were finally made, were generous to your school and made possible the operation of our facilities in the manner that you wanted them to be operated. It was most heartwarming to have the full support of the Society behind our requests, and I appreciate that it was this full support that in large measure impressed the members of the legislature with our need for increased appropriations.

For your help we are all most grateful, and for the forthright support of the legislature we are all most appreciative. This was a convincing demonstration that our state means to continue to operate a medical school that will be a front-runner. This demonstration, of course, maintains the enthusiasm of all of us at a high level.

Society Committee. While the legislature was in session the executive faculty had the pleasure of meeting twice with the Society's Committee on Medical Schools. Dr. Whitley and his group were the first such to visit the school in many years. They were given a complete survey of the operations of the medical center and a résumé of its problems and future plans. This year we will be meeting again at approximately the time you will be reading this report. One of the high spots of the last year for all of us was the warm vote of confidence extended to your medical school by this committee in its report that was accepted by acclamation by our House of Delegates.

Admission of Students. We have just concluded our screening of applicants for admission for this year's entering class and have accepted 100 students of the approximately 300 who applied. It became apparent when we visited with the medical school committee last year that many of you would like to know more about how the admissions committee makes its selections. In brief, we base our decisions upon these factors: previous scholastic attainment, results of the medical college admission test, results of personal interviews, and letters of recommendation.

We base a great deal of our decision on scholastic achievement because the records show that it is a rare individual, indeed, who serves with great distinction as a medical student after a low collegiate scholastic record. In cases of doubt, where scholastic achievement happens to be borderline, we place emphasis on the candidate's record in the medical college admissions test. If the record here is good, we are influenced in the candidate's direction.

The two areas of greatest difficulty are the interview and the letters of recommendation. Let me first say a word about the latter. We have a difficult time in obtaining what we consider to be thoughtful letters of recommendation. Unfortunately, the great majority of our letters are glowing and contain little solid comment. If each of our students were the paragon of virtue, honor, and intelligence that the letters indicate him to be, we would be teaching annually a new company of saints. Such unfortunately is not the case.

The personal interview is perhaps the one place where the unfortunate personality is revealed. Our interviewing technique is far from perfect, but we believe that interviews help us to screen out applicants unfitted emotionally or personality wise for the study of medicine.

We look upon our mission not as providing large numbers of doctors for Kansas but rather as providing the *best possible doctors* for Kansas. We are training those that we believe will be physicians whom you and we can accept proudly as members of our profession and to whom the patient will be able to go in confidence, confident of their knowledge and their integrity.

At the present time all of the medical schools in the United States are faced with a reduction in the number of qualified applicants. We are no exception. This lack of applicants is related to the low birth rate of the depression years. We are now at the nadir; in a few years the numbers will increase again, and we shall be faced with the necessity of turning down qualified applicants. At the present time we are encouraging young people to the study of medicine knowing full well that if their academic records are good and their personalities acceptable they will be able to gain admission easily.

Personnel. Since our last report we have added personnel in teaching positions in many of our departments. New teachers have joined us in the Departments of Psychiatry, Radiology, Medicine, Pediatrics, Surgery, Pharmacology, Pathology, and Obstetrics. Additionally, following the retirement of Miss Woodruff after long years of faithful service, we obtained the services of Mr. Cavanagh, our new librarian.

There are two new appointments of which I should like to make more than passing mention, not to slight the other excellent new people but to point up new developments. Last year, the Kansas Tuberculosis and Health Association endowed the Christmas Seal Chair of Thoracic Diseases for a five-year period. This endowment marked a significant advance for the association and for the medical school. From the standpoint of the association, it represented the affirmation of the fact that education is a prime mission of all health agencies. From our standpoint, it was most significant in that it is the first endowed chair in the medical school. We look to the day when there will be many more. Dr. Martin Fitzpatrick is the first appointee to this endowed chair.

In September of last year, Dr. Vernon Wilson joined us as Assistant Dean in Charge of Student Affairs. This most important position he has filled admirably. Student morale is at a high level, and this is in large measure a tribute to him. Our students are our greatest responsibility, and we mean them to have the finest of advice and counsel. This position will be a permanent and increasingly important one.

Physical Plant. We are now beginning to occupy fully the new building housing hospital services and the beds for chest diseases. For the first time we have an adequate obstetrical service geographically complete. Additionally, we have a completely equipped nursery for premature infants as well as a modern facility for the treatment of chest diseases. The new cafeteria is approaching completion, and we hope to occupy it within the next six weeks.

Of paramount importance to all of us is the Student Union-Continuation Study Center which is now almost completed. A director for the building has been employed, and we are looking forward to its early use. We have scheduled its dedication for April 4 and intend to open it then, despite the fact that we do not have funds sufficient to complete its furnishing.

You have been most generous in your response to our pleas for funds, and we are grateful to all of you who contributed so generously. This building represents a contribution of at least $50,000 from physician alumni of the university and members of the Society. Contributions this year to the American Medical Education Foundation have been the highest of any year to date. Your continuing support to this foundation is vital to us, and we are delighted to see contributions to it continually increasing.

Our psychiatry building is now in final drawing stage, and construction on it should begin before July. This represents the last building made available by the Rural Health Program passed by the legislature in 1949.

Postgraduate Courses. Postgraduate education, which has always been a co-operative venture between the Kansas Medical Society and the medical school, continues to be an ever-increasing program. We are proud of our position in the forefront of this field. Rather than hold the line we intend to enlarge this program and offer more courses for you. Your suggestions along this line continue to be most helpful, and we solicit them.

Need for Library Unit. The greatest need that we have at the medical center is for a new library. The present one is cramped, has seats for only 60 of the 1,000 people who must use it, and has no space into which it can expand. Further, the weight of the books is a constant threat to the structural soundness of the administration building which was not constructed to withstand such weight. For these reasons we are going to request the next legislature to appropriate from the Educational Building Fund sufficient funds to erect a two-story library unit. This is the only building request that we will advance.

Your medical school continues to attract national attention and to increase in stature. All of this is a tribute to your continuing support and enthusiasm for it. I am looking forward to many more years of close co-operation—in my dual position as director of your medical center and member of the Kansas Medical Society.

Kansas Medical Day

The annual Kansas Medical Day was held at the University of Kansas School of Medicine on Monday, March 8, 1954. In charge of the program was Dr. Lucien R. Pyle, Topeka, president of the Kansas Medical Society, and the audience consisted of 225 junior and senior students.

The program included opening remarks by Dr. W. Clarke Wescoe, dean of the school, and Dr. Mahlon H. Delp. Mr. Carl C. Lamley, superintendent of Stormont-Vail Hospital, Topeka, and president of the Kansas Hospital Association, spoke on the physician's responsibility in the hospital. The continuing interest by the medical board in the practicing physician was discussed by Dr. Murray C. Eddy, Hays, a member of the Kansas State Board of Medical Registration and Examination and president-elect of the Kansas Medical Society. Blue Cross-Blue Shield as a phase of prepaid health insurance was discussed by Dr. Henry S. Blake, Topeka, president of Kansas Blue Shield, and by Mr. Proctor Redd, Topeka, director of professional and public relations.

Mr. Albert M. Ross, Kansas City, examiner for Workmen's Compensation, discussed the physician's duties and responsibilities under the Workmen's Compensation program. Mr. Ernest H. Hall, Kansas City, Missouri, narcotic agent, spoke on the physician's responsibility toward complying with the requirements of the Harrison Narcotics Act. Services of county and state medical societies to physicians were described by Mr. Ray Selbach, executive secretary of the Shawnee County Medical Society, Mr. H. Martin Baker, executive secretary of the Sedgwick County Medical Society, and Mr. Oliver E. Ebel, executive secretary of the Kansas Medical Society.

A coffee period in the Student Union Building concluded the day's activities.

Cancer Conference in Wichita

Arrangements have now been completed for the Sixth Annual Mid-West Cancer Conference to be held at the Broadview Hotel, Wichita, April 1 and 2, 1954. Although the program is primarily scientific, a social event will be included—a banquet the evening of April 1 to which both doctors and their wives are invited. Dr. Cornelius P. Rhoads, director of Memorial Hospital, New York City, will be the banquet speaker.

Biographical notes on several of the guest speakers were published in the January and February issues of the Journal. Information on other guest lecturers is given below.

Dr. A. N. Arneson, St. Louis, is professor of clinical obstetrics and gynecology and associate professor of clinical radiology at Washington University School of Medicine. He received his medical degree from the same school in 1928. Dr. Arneson is a diplomate of the American Board of Radiology and of the American Board of Obstetrics and Gynecology and is a fellow of the American College of Radiology. He is a member of the Radiological Society of North America and of the American Radium Society.



Dr. Hugh F. Hare, Los Angeles, is associated with the Los Angeles Tumor Institute. He was graduated from Harvard Medical School, Boston, in 1928. In addition to being a diplomate of the American Board of Radiology, he is a fellow of the American College of Radiology and a member of the Harvey Cushing Society, American Roentgen Ray Society, Radiological Society of North America, and American Radium Society.

Dr. N. L. Higinbotham, New York City, who is associate clinical professor of surgery at New York Medical College, is a graduate of McGill University Faculty of Medicine, Montreal, with the class of 1926. He is a diplomate of the American Board of Surgery and a fellow of the American College of Surgeons.

Dr. Higinbotham is also assistant clinical professor of surgery at Cornell University Medical College.

An important member of the staff of the Overholt Thoracic Clinic, Brookline, Massachusetts, who will take part in the program is Dr. Richard H. Overholt. He is also on the staff of the New England Deaconess Hospital. Dr. Overholt was graduated from the University of Nebraska College of Medicine in 1926. His memberships include the New England Surgical Society, the American Association for Thoracic Surgery, and the American Roentgen Ray Society. He is a diplomate of the American Board of Surgery and a fellow of the American College of Surgeons and of the American College of Chest Physicians.



The conference is being sponsored by the Kansas Division of the American Cancer Society and the Committee on Control of Cancer of the Kansas Medical Society. All physicians in Kansas and adjoining states are invited to attend. No registration fee will be charged.

Good public relations for the medical profession begin in the office of each individual doctor.

K.M.S. annual meeting, May 2-6, 1954, Topeka.

BLUE SHIELD

Sponsorship on the Local Level

The new Blue Cross-Blue Shield non-group enrollment seems to be off to a fine start. Campaigns were completed in nearly 25 counties during January and February, and in many counties results have been even more successful than had been anticipated.

More than ever in this year's new program, the importance of participation or sponsorship by the medical profession at county medical society level has been emphasized. It is only natural that the prospective members feel a closer relationship with Blue Cross-Blue Shield when they can be assured their own physician and local hospital are directly connected with the program.

what can be done

There are many ways that members of county medical societies can co-operate in the community enrollment program. They can sponsor endorsement advertising in the local newspapers; they can announce in public news stories that they have pledged their co-operation; they can mail literature with their billings to patients prior to the enrollment; they can use their offices as information centers, and keep their assistants well informed on the program in order that they can answer questions about Blue Cross-Blue Shield services.

When the county medical society has agreed to participate in enrollment, literature and material will be furnished by the Blue Shield office. Members of the Blue Cross-Blue Shield staff are presently appearing at county medical society meetings and medical assistants' meetings to explain the new program in all of its aspects.

The Douglas County Medical Society might well be cited as one of the organizations in the state which has participated in practically all of the projects listed above. The society as a whole served as the sponsoring organization for the county-wide enrollment; Dr. J. W. Campbell, president of the society, was the chairman of the campaign, giving the enrollment a definite local tie-in.

The society sponsored a paid newspaper advertisement announcing the campaign. Dr. Penfield Jones, long active in the Blue Shield program, a medical society councilor, and president of the Kansas State Board of Health, appeared on a 15-minute radio interview over station KLWN at Lawrence in which he explained the services of Blue Cross-Blue Shield. Dr. G. E. Manahan, Blue Shield Relations Committee Chairman from District II, also played an important role in the program.

Enrollments to be held in March include Bourbon, Ellis, Finney, Franklin, Jewell, Marion, Marshall, Pratt, Reno, Smith, and Thomas counties.

Nominations for 1954-1955

In accordance with provisions of the Constitution and By-Laws, the Nominating Committee of the Kansas Medical Society announces here the names of candidates who will be listed on the ballot for the annual election to be held on May 6, 1954. Additional nominations, of course, may be made from the floor.

Because some of the members of the Kansas Medical Society might not be personally acquainted with each of the physicians nominated for office, the committee submits the following brief data.

For President-Elect

Dr. John M. Porter, Concordia, has served as secretary and second vice-president and is currently first vice-president of the Kansas Medical Society. He has also been delegate to the American Medical Association and president of the Kansas Heart Society.

For First Vice-President

Dr. Clyde W. Miller, Wichita, has been a councilor and is currently second vice-president of the Kansas Medical Society. He was chairman of the Committee on Public Relations and was the first president of the Kansas Academy of General Practice.

For Second Vice-President

Dr. Conrad M. Barnes, Seneca, besides numerous offices in his local society, has been a member of the Dean's Advisory Committee for the School of Medicine at the University of Kansas and has been president of Kansas Blue Shield.

Dr. Louie J. Beyer, Lyons, has long been an active participant in Kansas politics and has served as a national committeeman from this state. Besides offices in his local society, he has served six years on the Council of the Kansas Medical Society.

Dr. Thomas P. Butcher, Emporia, has held numerous offices in his county society, including its presidency. He has been chairman of the Kansas Medical Society Committee on Allied Groups and has served on other committees.

Dr. Dwight Lawson, Topeka, besides numerous offices in his county medical society, has served as president of Kansas Blue Shield and is a member of the Editorial Board.

Dr. John W. Randell, Marysville, besides numerous offices in his county society, has been a member of the Council of the Kansas Medical Society and for the past several years has been chairman of the Committee on Endowment.

For Constitutional Secretary

Dr. James A. Butin, Chanute, has held numerous offices in his county medical society and has been active in the work of the district society of the nine-county area comprising the third councilor district.

Dr. Karl E. Voldeng, Wellington, besides numerous offices in his county society, has been chairman of the Committee on Control of Cancer and is now president of the Kansas Division of the American Cancer Society.

For Treasurer

Dr. John L. Lattimore, Topeka, has been an active participant in Kansas politics, having served four terms in the legislature. He has been president of the Kansas Medical Society and is currently treasurer.

For A.M.A. Delegate 1955-1956

Dr. Laurence S. Nelson, Salina, has been president of the Kansas Medical Society and is currently delegate to the American Medical Association. He has also served the state as a member of the Kansas State Board of Health.

For A.M.A. Alternate 1955-1956

Dr. Lucien R. Pyle, Topeka, has been editor of the Journal and was formerly chairman of the Volunteer Advisory Committee for Kansas. He is currently president of this Society.

Dr. Frederick E. Wrightman, Sabetha, besides serving his county society in various capacities, has been a member of the Legislative Committee and has served as a Kansas representative of the A.M.A. Committee on Legislation.

Annual Meeting, May 2-6, 1954

Committees in charge of the annual meeting of the Kansas Medical Society to be held at Topeka, May 2-6, report all is now in readiness. The program has been arranged, social events have been scheduled, and specialty societies are going ahead with plans for separate meetings.

In general, the outline of former years will be followed, although one change has been made in the schedule for scientific sessions. During each morning session on Tuesday, Wednesday, and Thursday, guest speakers will present papers. The afternoon sessions on Tuesday and Wednesday will be given over to panel presentations, two each day on different subjects. Guest speakers will also participate in the panels. Separate sessions, as usual, will be held for physicians engaged in eye, ear, nose, and throat practice.

The following speakers from out-of-state will present the program:

David D. Akers, M.D., Denver, Colorado
Frederic Burke, M.D., Washington, D.C.
James R. Cook, M.D., Cleveland, Ohio
Oliver Cope, M.D., Boston, Massachusetts
Charles E. Iliff, M.D., Baltimore, Maryland
Ormand C. Julian, M.D., Chicago, Illinois
Andrew A. Marchetti, M.D., Washington, D.C.
Paul M. Moore, M.D., Cleveland, Ohio
Shepard Shapiro, M.D., New York, New York
Maurice Stauffer, M.D., Rochester, Minnesota
Bernard A. Watson, M.D., Clifton Springs, New York

Complete information on the annual session will be published in the April issue of the Journal.

Party Honoring Mrs. Schaefer

A party in honor of Mrs. Leo J. Schaefer, president of the Woman's Auxiliary to the American Medical Association, was given by the Saline County Medical Society at the Salina Country Club on February 3. The guest list included all officers and councilors of the Kansas Medical Society and of its Auxiliary.

Dr. Franklin D. Murphy, chancellor of the University of Kansas, made the address of the evening. The program included a reception, dinner, and dance.

Mrs. Schaefer is the first Kansan to hold office in the women's national organization.

Although medical social work is a relatively young profession, there are approximately 3,825 persons actively engaged in this work in the United States today, according to a recent release from the U. S. Department of Health, Education, and Welfare. There are, however, three times as many positions open in this field as there are persons to fill them, and it is estimated that 800 to 1,000 graduates a year will be needed to fill vacancies.

The largest number of social workers, approximately 41 per cent, are located in the northeast section of the country. Three-fourths of the total are employed in hospitals and clinics, while the two next largest groups work with the physically handicapped and other special groups. Most medical social workers are women, 93 per cent, although the number of male graduates from social work courses is gradually increasing.

Attend the annual meeting, Kansas Medical Society, Topeka, May 2-6, 1954.

Parotid Tumors

Tumor Conference

Edited by Chauncey G. Bly, M.D., and Bernard L. Klionsky, M.D.

Dr. Helwig: Today we have a group of three patients with tumors of the parotid gland. The first two cases illustrate some of the usual difficulties encountered in dealing with these tumors. The last case poses current difficult problems in both diagnosis and management.

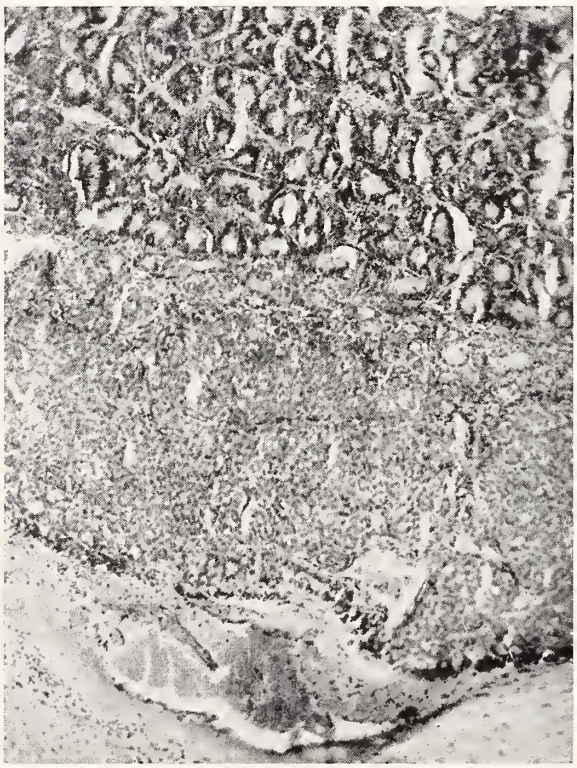


Figure 1. Benign parotid adenoma (case 53-99). This area shows the focus of transition from the typical acinar or duct-like structures above, to solid sheets of poorly differentiated atypical cells, still enclosed by the fibrous capsule seen at the bottom. X 82. Hematoxylin and eosin.

CASE NO. 53-99

Dr. Saffier: This 54-year-old white male states that he had mumps five years ago. The swelling or mass did not subside, however, but lay dormant until one year ago, when some local discomfort began to appear. X-ray films of the area revealed no stone or obstruction of Stensen's duct. The tumor mass was biopsied here and two days later was completely removed.

Dr. Boley: In both the biopsy and the final operative specimen, the tumor is well enclosed by a capsule, showing hemorrhage and edema of operative origin. The tumor itself is composed primarily of columnar cells forming uniform duct-like structures or acini with scant supporting stroma. We are impressed by the high degree of cellularity of the tumor and by the few small areas of necrosis.

One of the final sections (Figure 1) shows a focal transition from duct-like arrangement of columnar cells to sheets of solidly packed paler polygonal cells. Other areas show slight nuclear variation. In one area, a few acini or collections of cells are seen within the fibrous capsule. Despite these occasional atypical features, we cannot call the tumor an adenocarcinoma since it is so well encapsulated. Our final diagnosis was and is: parotid adenoma with atypical changes and local infiltration of the capsule, meaning that this patient should be carefully followed.

Dr. Robinson: This case illustrates some of the operative difficulties encountered in dealing with parotid tumors. A large mass, 3 cm. in diameter, was found to be tightly wedged into the neck tissues below the angle of the mandible. The tumor had apparently arisen in the retromandibular part of the parotid and grown forward, displacing the normal part of the gland and stretching and spreading out the branches of the facial nerve. Since the biopsy indicated this to be a benign parotid adenoma, we preserved the facial nerve by tediously dissecting each of the branches completely free and removed the tumor from below. When we were through, the patient had a facial nerve which was totally intact anatomically, but not functionally. It has now been five months since the operation, and, as you can see, the patient's nerve function has been considerably restored. He can wrinkle his forehead and eyelids, but there is still some facial droop and asymmetry on smiling. However, we expect all of this residual dysfunction to return to normal within the next few months.

CASE NO. 53-100

Dr. Saffier: This second patient is a 44-year-old colored man who has a mass in the right side of the neck. The tumor had grown steadily and slowly over 1½ years, but produced no symptoms.

On examination, a smooth, firm, movable, well circumscribed, non-tender subcutaneous mass about

Cancer teaching activities at the University of Kansas Medical Center are aided by grants from the National Cancer Institute and the Kansas Division of the American Cancer Society.

Dr. Bly is a Scholar in Cancer Research of the American Cancer Society. Dr. Klionsky is a Trainee of the National Cancer Institute.

6 cm. in diameter was felt over and behind the ramus of the right mandible. The bulk of the tumor appeared to be in the anterior superior triangle of the neck. The remainder of the physical examination was negative. Our clinical impression was mixed tumor of the parotid.

At operation a firm, encapsulated mass deep in the parotid gland was removed by sharp dissection during which the capsule was inadvertently opened. One week after the surgery there was still some firm swelling in the operative area with some evidence of persistent right lower facial weakness.

Dr. Boley: This tumor is lobulated but does not have the cartilaginous, slimy appearance which many mixed tumors of the parotid have. By gross and microscopic examination, the tumor is fairly well encapsulated, although the thickness of the capsule varies markedly from area to area. The tumor generally is more cellular than are most mixed tumors. The slight palisading of nuclei seen in some of the most cellular

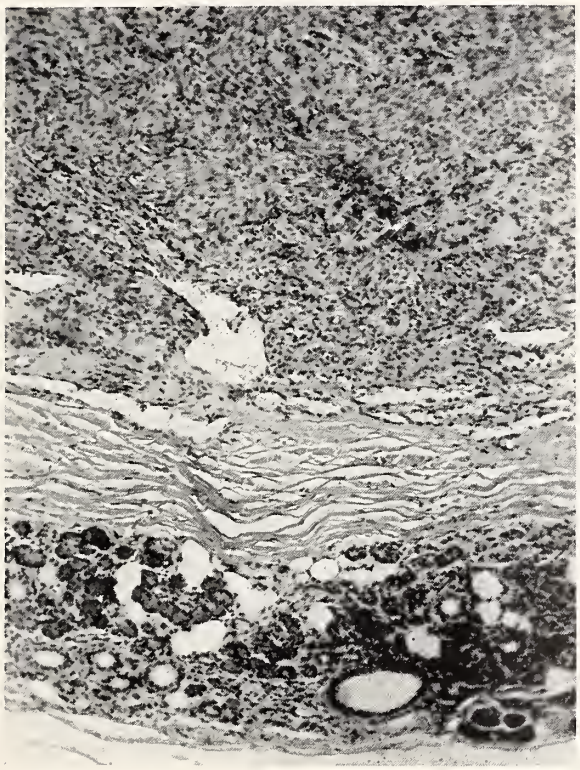


Figure 2. Benign mixed tumor of parotid (case 53-100). This unusually cellular area containing spindle-cells with slight nuclear palisading, and minimal stroma is separated by thick fibrous capsule from the slightly compressed normal parotid acini and ducts below. X 47. Hematoxylin and eosin.

areas (Figure 2) is not so marked as in the Verocay bodies seen in neurogenic tumors. In some areas, there are cribriform or cylindromatous patterns with much mucoid stroma (Figure 3). In other areas definite acini and hyalinized stroma are found. No mitoses, marked nuclear variations, or frank invasion of blood vessels are seen. This is a benign cellular mixed tumor of the parotid. However, the tumor is likely to recur, particularly with the break in the capsule during surgery.

Dr. Helwig: It seems that we have already opened up two fields of discussion—problems in operation and in diagnosis. Dr. Robinson, would you elaborate

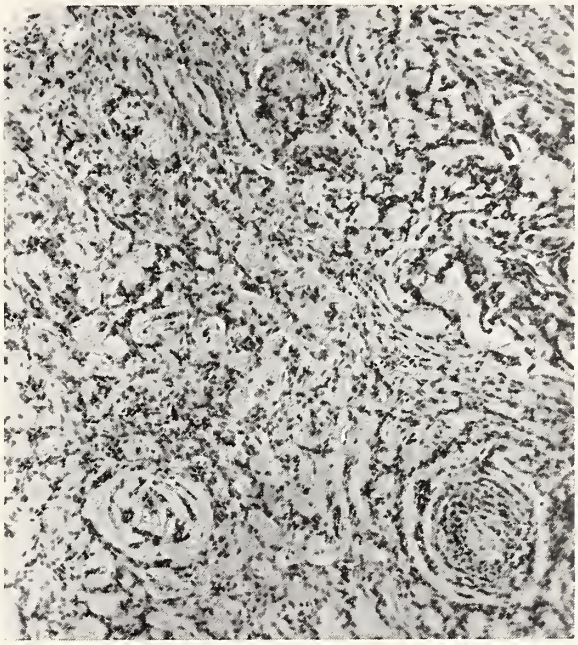


Figure 3. Benign mixed tumor (also case 53-100). This more usual area of the tumor shows less cellularity and prominent muco-hyaline change in the stroma. X 82. Hematoxylin and eosin.

on some of the factors which concern you in the general treatment of the patient with a benign tumor of the parotid.

Dr. Robinson: Certainly the treatment of choice for adenomas or benign mixed tumors of the parotid is surgical removal. In the sphere of technical problems, the main operative and postoperative difficulties which arise are related to the facial nerve. Much too often in the past, over-conservative operations such as simply shelling the tumor out of its pseudocapsule, have been done primarily in order to save the nerve. One must, however, balance nerve preservation against chances of tumor recurrence.

Thus, one must first decide how much of the normal parotid gland should be removed with the tumor. Although surgeons differ in recommendations on this point, we like to have a cuff of normal parotid tissue 3 mm. thick all around the tumor capsule. This is not always possible if one wants to leave the facial nerve intact. However, in almost all cases of benign parotid tumors, the nerve branches can actually be dissected out without causing permanent damage. Even when one takes off a cuff of normal parotid around the tumor, including the capsule or pseudocapsule, one

will have some recurrences. These must be considered as more than just multicentric origin of the primary tumor.

It is much harder of course to do the proper operation, removing a cuff of normal tissue with the tumor. There are no good planes of cleavage, and one must dissect each millimeter very carefully with knife or scissors, testing as he goes, to make sure that he is not making the face twitch. The entire face must be draped out and someone assigned just to watch the face all the time.

The difficulty in continuous observation of the nerve function is one reason these benign tumor excisions cannot be done as out-patient procedures. Using local anesthesia, one cannot tell whether the nerve is capable of working or not when it is directly stimulated. Even after the operation, the local anesthetic effect is maintained enough that one still is unable to tell whether the nerve is working well. Furthermore, since local blocking of the nerves is not technically good enough for adequate anesthesia, it is necessary to field infiltrate. During this latter procedure, the tumor itself may be inadvertently punctured by the needle, thus increasing the chances for tumor recurrences.

Dr. Boley: I would like to know how disturbed Dr. Robinson is when he discovers that the patient's face is paralyzed the day following operation. A less experienced surgeon might become worried at such a finding.

Dr. Robinson: No surgeon likes to see his patient grimacing, or drooling, or talking from the side of his mouth as a result of an injury to the facial nerve. If I know that I have left the nerve anatomically intact, that I have dissected practically all of the soft tissue, and that the blood supply has been undisturbed, I expect that normal function will eventually return. Most will function normally as soon as any postoperative edema disappears. Few show even as moderate a defect as does the first patient we showed, but even in those, function will eventually return.

Dr. Tice: In what percentage does surgery result in damage to the facial nerve? How hard is it to find the facial nerve during operation?

Dr. Robinson: I have not looked up our statistics recently, but I should guess that there has been permanent damage to the facial nerve in much less than 10 per cent of cases operated on for benign parotid tumors. By permanent, we mean damage in which there has not been recovery in a few months' time, since it may take up to 18 months for complete regeneration of the facial nerve fibrils along the axon sheath after functional damage. It is not as hard as one might think to find the facial nerve. It has long been know that a branch of it will be invariably found if one follows the communicating posterior branch of the facial vein up to the lower pole of the parotid and into the gland. This branch of the facial nerve can then be traced to the main trunk.

Dr. Helwig: The second broad topic for discussion is the problem of diagnosis of parotid tumors. Dr. Boley, I know you have some general feelings on this subject.

Dr. Boley: A specific diagnosis on a given parotid tumor is sometimes difficult for the pathologist to make. There are many different and some elaborate classifications, and some tumors seem to defy any of these classifications. The term "mixed tumor" is widely used on the basis that this type of tumor contains a mixture of epithelial elements and stromal or mesenchymal elements, both showing neoplastic changes. About two-thirds of all parotid tumors are classified under this name. However, the more areas the pathologist studies from each parotid tumor, the more different types of epithelial and stromal change he will find in each tumor. Moreover, as illustrated in our first two cases today, atypical changes are not infrequent. Willis[1] goes so far as to state that both the concept and the name, "mixed tumors," are misuses since he feels, in all of these types of tumors, that only the epithelium shows neoplasia and that the stromal changes are secondary and incidental. Instead of designating this type as mixed tumor, he would use the terms pleomorphic adenoma or adenocarcinoma, depending on whether the tumor is benign or malignant.

Dr. Robinson: There are several useful points to keep in mind when arriving at a clinical diagnosis, before biopsy or excision. It's fairly easy to say that those parotid tumors which to palpation are quite hard and firmly fixed are malignant, but they are not always malignant. We have found them sometimes in anatomical positions where they are so tightly squeezed by growth between bony parts that they are apparently clinically fixed, but are not really fixed. For example, a few, growing between the angle of the mandible and the mastoid, are so tightly bound as to give the impression that they are malignant and will necessitate radical surgery. However, one often finds during the operative procedures that they are not fixed, but can be completely removed.

Generally, parotid tumors that cause pain are malignant. Tumors that involve the facial nerve to such an extent that the nerve does not function well preoperatively are usually malignant. Factors of sex or race are not important diagnostically.

Mixed tumors of the parotid gland can occur at any age, but they usually occur between the ages of 20 and 40 years. The patient himself may accidentally feel the tumor for the first time, or have the swelling brought to his attention by someone else. Mixed tumors grow quite slowly, usually without any symptoms or signs other than the swelling or enlargement. Carcinomas of the parotid gland tend to grow more

quickly, occur in older age groups, cause pain, and are stony hard.

Dr. Johnson: Usually the main thing that a surgeon would like to know before operation is whether he is dealing with a relatively benign or with a malignant tumor, since the management is different. There are tumors that clinically seem obviously benign, while others seem obviously malignant. There is always a smaller group between, in which the clinical impressions are uncertain. In some of these latter cases, needle aspiration biopsy has been done preoperatively in the hope that we as pathologists could differentiate the benign from the malignant lesions. I must confess that in only relatively few instances has an aspiration biopsy helped us to make a decision. We are continuing to evaluate this procedure, however.

Dr. Robinson: Although the surgeon occasionally will do an open biopsy of the parotid, there is the one real danger previously mentioned. If the tumor is surrounded by a pseudocapsule, there are chances that, by breaking it, one will contaminate the operative field with tumor cells, whether benign or malignant, which may then be possible foci of recurrence. Therefore, we do not like to do a biopsy just before starting one of these operations.

One point of help during the exposure of the tumor is that if one sees tumor growing around nerves, he can be reasonably sure that it is not a benign tumor grossly. Sometimes I need to call Dr. Boley to observe the gross tumor and to do a rapid frozen section of the tumor to find out microscopically what we are dealing with, so that I can decide the extent of operative procedures necessary. Such was done in the first case discussed today, where some of the nerve branches appeared almost to be imbedded in the surface of the tumor bulging up from behind the mandible; frozen sections showed the tumor to be benign and everywhere enclosed by fibrous capsule.

The proper treatment of a benign parotid tumor has already been discussed. Whenever one is sure that he is dealing with a malignant tumor, he must forget conservatism and nerve preservation and do radical tumor surgery, even though this may necessitate removal of the entire area. For example, one may have to pick up and divide the trunk of the facial nerve, chisel off parts of the mandible and the mastoid bones, and remove the ear canal along with the tumor, if necessary. Even if such extensive surgery is performed, the chances of salvage with an advanced malignancy are very poor. One should not, however, give up completely, or he will lose all of his patients with advanced carcinoma, including the small salvageable group.

CASE NO. 53-101

Dr. Saffier: This third case is a 7½-year-old boy who came in because he had a swelling of the left parotid area of three months' duration. When we first saw him, the tumor growth was lobulated and about 3 cm. in diameter, appearing grossly rather typical of the mixed tumor type. Upon exploration, the tumor still looked superficially like a mixed tumor. However, as the dissection was carried anteriorly, it was noted that the tumor had involved branches of the facial nerve and invaded the masseter muscle.

Dr. Boley: In this case, adipose tissue and fibrous tissue blend in with tumor tissue, with no true capsule to separate tumor from parotid gland or muscle. Most microscopic fields of the tumor show clumped and free larger rounded cells intermingled with irregular bands of spindle-shaped elongated cells with possibly some small processes. Some mitotic figures are seen. Special stains showed that this tumor is forming some reticulin with no set pattern, although it tends to surround occasional groups of cells or single cells. This production of some reticulin suggests that this tumor may be a sarcoma rather than a very anaplastic carcinoma of the parotid gland.

Dr. Robinson: From the moment we saw the gross involvement of masseter muscle and twigs of the facial nerve, we felt that this boy's prognosis was poor. Even though we were able to get fairly well around the tumor, the gross and histologic appearance of the tumor seems to preclude further radical surgery in a boy of this age.

I asked Dr. Tice to see him with us. He gave him a course of x-ray therapy, feeling that since this was an anaplastic tumor it might respond favorably. We saw the boy again three days ago and felt some small shotty nodes in the neck. These might represent merely reaction from the operation and radiation therapy, although they may have more serious import. We will probably watch him a little while before doing anything more. Certainly if these nodes contain metastases, further surgery will probably be of little value.

Dr. Tice: We gave this boy about 2,000 roentgens to the localized area and to the neck glands, a dose which will ordinarily kill a highly anaplastic tumor. We have to keep in mind also the growing bones in the area. For example, we will certainly have already damaged that growing mandible with the dose that we have given. If he survives, he will also have trouble with his teeth and may lose them. On the other hand, he might still have tumor present from which he will die if we do not give him further heavy doses of radiation. The problem we face is how far we dare treat him in the future if necessary.

Dr. Robinson: Malignant tumors are not the only ones in which we must decide whether any further treatment is indicated. We saw in the clinics today a man who has swelling in the left parotid gland region, which becomes more prominent when he eats or drinks. There is a slightly tender freely movable but indiscrete firm thickening in the belly of the

masseter muscle. This mass has been present for about 7 years.

Two years ago he had pain in the left cheek. After dissecting through some of the fibers of the masseter muscle, we found a 1.0 cm. nodule which popped out of its capsule. Dr. Boley, recalling one parotid tumor in which the entire center was infarcted, was unable to find any tumor cells even around the periphery and called this an organized hematoma.

We have not tried to visualize any possible obstruction in Stensen's duct. Dr. Tice tells me that either Lipiodol or Diodrast would work quite well. Until we have more evidence that this mass might be a tumor, rather than just fibrous tissue with partial duct obstruction, no further treatment seems indicated. If we do more surgery, we are almost sure to damage the facial nerve because of technical difficulties related to the scar tissue from the previous operation and inflammation in the area.

Dr. Helwig: As we look back over parotid tumors in general, I would like to re-emphasize that there is still that small group of tumors where, even with the whole tumor specimen to examine, the pathologist cannot be too sure of the relative benignancy or malignancy. Fortunately, that group is relatively small. To help us, we now have the article by Foote and Frazell.[2] This is one of the finest articles I have ever seen on tumors of the parotid, based on patients studied over a 20-year period at Memorial Hospital in New York. In their group of 877 cases of salivary gland tumors, 551 (63 per cent) were mixed tumors, and only 57 of those were malignant. This figure corresponds roughly with that reported by most people, 10 to 15 per cent of mixed tumors being malignant. As one studies carefully the photomicrographs in this article, he may wonder whether some of the tumors shown should have been called benign or malignant. However, he must bow to the very excellent follow-up on actual behavior which supports the decisions of these authorities.

Dr. Robinson: In the 25 or so cases of mixed tumors of the parotid that we have seen here in the past five years, there seems to be a much higher percentage of malignancy, around 40 per cent, than in other larger series. This may be a reflection on the smaller size of our series.

Dr. Helwig: Are they really malignant, or are they merely called malignant because they recurred?

Dr. Boley: No, these figures on malignant mixed tumors are based upon histological criteria similar to those of Foote. Moreover, since 65 per cent of the salivary gland tumors seen here are mixed tumors, and 12 per cent are mucoepidermoid carcinomas, about the same ratios as seen in Foote's much larger series, we do not appear to be confusing these two types of tumor.

Dr. Robinson: I do not think that, in our concluding remarks, we can over-emphasize the problem of recurrences, so characteristic of mixed tumors of the parotid. Even if one is sure that the tumor is benign, he should not simply shell it out from its capsule or he may expect to find recurrences in up to 40 or 50 per cent of his cases. Attempts to preserve the facial nerve in operations on malignant mixed tumors will also result in a high percentage of recurrences since tumor cells will usually be left behind with the nerve. Fortunately, metastases from malignant mixed tumors are uncommon, and recurrences form the major problem.

Dr. Boley: In the past, many of the recurrences were accounted for by assuming the primary tumors to be of multicentric origin. Foote[1] makes the statement now that in his group of 551 mixed tumors, he has never seen one of multicentric origin. Only in those tumors that had previously been operated upon have there been multiple nodules. Sometimes as many as 8 or 10 nodules had apparently arisen from seeding during inadequate surgery or interference, producing thereby a multinodular tumor. He believes that there are fewer than five well documented cases in which multiple primary nodules have been found in mixed tumors of the parotid gland.

Dr. Joffe: How long does it require for a benign tumor to recur after inadequate excision?

Dr. Robinson: One of the difficult things about evaluating surgery of parotid tumors is that the recurrences come very slowly and insidiously. For that reason, one should follow these patients at definite intervals to evaluate critically the problem of recurrence. For the most part, we do not find recurrences in less than 6 months, but we do find them on an average of one to two years after excision. Sometimes recurrences appear after 4 or 5 years or rarely even later than that. Many surgeons feel that if 5 years have gone by without recurrence, the patient has been cured. We have had one patient who had a recurrence of a cylindromatous growth of the submaxillary gland after 12 years.

On the other hand, if a benign mixed tumor should recur and be inadequately treated a number of times, the tendency is for each recurrence to look a little bit different and more malignant than the preceding recurrence.

Dr. Helwig: Mixed tumors to me are either predominantly cylindroid or predominantly mesenchymal. It has been my experience, and also that of the group at the Mayo Clinic,[3] that the more predominantly cyclindroid they are, the more tendency there is to local recurrence and invasion. One patient who had a cylindromatous type of mixed tumor simply shelled out of the hard palate, died five or six years later of meningeal involvement by recurrent tumor. On the other hand, I have been following a patient who is still alive and well after multiple excisions of re-

currences from a cylindromatous tumor removed from the tongue 47 years ago.

BIBLIOGRAPHY

1. Willis, R.: Chapter on epithelial tumors of the salivary glands; in Pathology of Tumors, 320-348, Butterworth, London, 1953.
2. Foote, F. W., Jr., and Frazell, E. L.: Tumors of the major salivary glands, Cancer 6:1065-1133 (Nov.) 1953.
3. Quattlebaum, F. W.; Dockerty, M. B., and Mayo, C. W.: Adenocarcinoma, cylindroma type, of the parotid gland; a clinical and pathological study of 21 cases, Surg. Gyn. and Obstetrics 82:342-347, 1946.

COUNTY SOCIETIES

A meeting of the Riley County Society was held in Manhattan on January 20. Dr. Donald L. Rose, chairman of the Department of Physical Medicine, University of Kansas Medical Center, spoke on "The Conservative Management of the Painful Shoulder."

Dr. J. R. Pritchard was named president of the Bourbon County Society at a meeting of the group held in January. Dr. L. P. Randles was elected secretary.

Dr. Mahlon H. Delp, associate professor of medicine at the University of Kansas Medical Center, addressed the Sedgwick County Society at a meeting at the Lassen Hotel, Wichita, on February 2. His subject was "Contraindication of ACTH, Cortisone and Relative Compounds."

A meeting of the Smith County Society was held at the home of Dr. Victor E. Watts, Smith Center, on January 28. An oyster supper was served. During the business session, the following officers were elected: president, Dr. Victor E. Watts; vice-president, Dr. Buford Hartman, Kensington; secretary, Dr. Hugh G. Woods, Smith Center; delegate to state meeting, Dr. Hartman; alternate, Dr. Lafe W. Bauer, Smith Center.

A tumor clinic was a feature of the February 1 meeting of the Shawnee County Society. Dr. A. A. Fink served as moderator. Dr. Robert Brooker spoke on lung tumors, and Dr. Robert P. Woods discussed brain tumors. A professional education film prepared by the American Cancer Society was shown.

Members of the Montgomery County Society assisted in a Blue Cross-Blue Shield enrollment campaign during February by designating their offices as information centers for those wishing data on the plans.

Dr. M. D. Atwood was elected president of the Edwards County Society for 1954. Other officers are: vice-president, Dr. R. T. Unruh; secretary, Dr. Frank G. Meckfessel; delegate, Dr. R. E. Schnoebelen; alternate, Dr. W. P. Stoltenberg. The society gave its approval to a suggestion of the dentists of the county that fluorine be used in the Kinsley water supply.

A meeting of the Harvey County Society was held on February 1 with representatives of Blue Cross-Blue Shield as guests. Guest speaker for the evening was an attorney, Mr. D. W. Wheeler, Marion, who presented a paper, "Malpractice and Supreme Court Suits and Problems of Experimental Medicine."

Dr. Earl E. Miller was elected president of the Crawford County Society at a meeting held on January 30. Dr. David J. Lyons was chosen as vice-president, and Dr. James H. Bena was named secretary-treasurer.

A meeting of the Wyandotte County Society was held at the University of Kansas Medical Center on February 16. For the scientific program, Dr. Herbert C. Miller spoke on "Respiratory Difficulties in the Newborn," and Dr. E. Grey Dimond discussed "Inferior Vena Cava Ligation for Intractable Right Heart Failure."

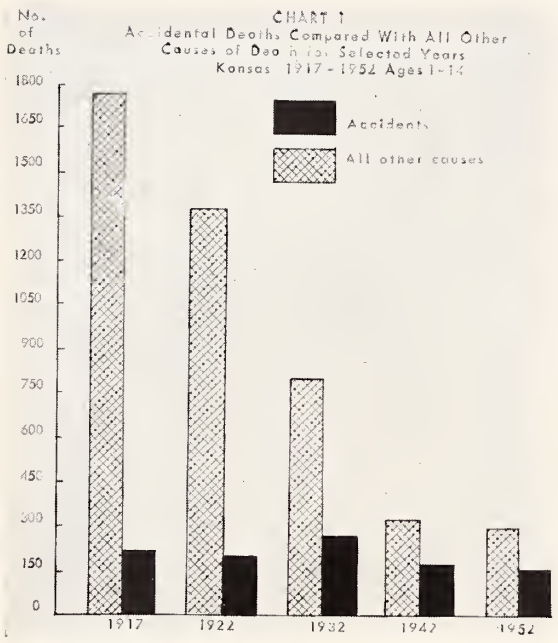
At a recent meeting of the Marshall County Society, Dr. John W. Randell was named president for 1954 and Dr. Henry H. Haerle was elected secretary.

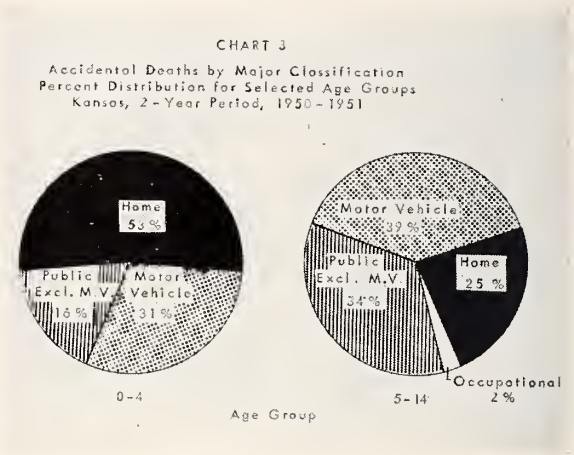
Dr. Clarence W. Erickson, Pittsburg, was guest speaker at a meeting of the Labette County Society held at the Parsons Country Club on February 10. His subject was "Hypertension."

Modern scientific discoveries have brought such great changes in medicines that 90 per cent of the physicians' prescriptions today could not have been written a scant 15 years ago. This great progress would seem to all but eclipse ancient methods of medication. But, on the contrary, the value and potency of many barks, roots, and leaves are so great they still are imported from far-off places for use as ingredients in some medicines.—*J. R. Anderson, chief pharmacognosist, Parke, Davis and Company.*

Accidents in Childhood

Prepared by the Committee on Child Welfare





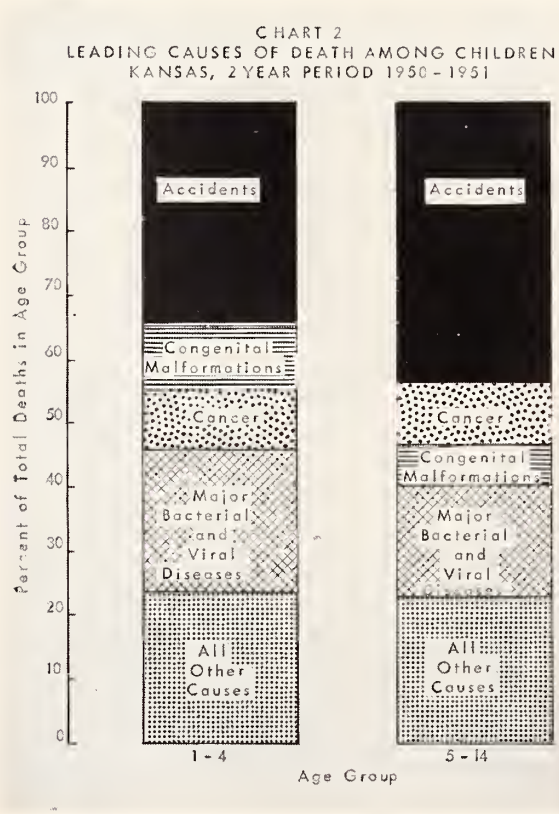


TABLE I

TYPES OF HOME ACCIDENTS BY 5 YEAR AGE GROUPS FOR AGES UNDER 15 YEARS
KANSAS 1952

	0-4	*5-9*	*10-14*
Burns, Conflagrations, Explosions	17	8	3
Suffocation, foreign objects	11	0	0
Mechanical Suffocation	7	0	0
Drownings	6	0	0
Poisonings, solids or liquids	4	0	0
Falls	4	2	0
Firearms	1	0	5
Electrocution	1	0	1
Poisonings, Gas	1	0	0
Other	6	0	0

Accidents in Childhood

Prepared by the Committee on Child Welfare

Along with immunizations, scientific nutrition, sulfonamides and antibiotics, and the science of child development, apparent changes have come in the relative importance of the various causes of childhood morbidity and mortality in Kansas as well as elsewhere.

As the other important causes of childhood deaths have been brought under control, the persistent accident toll in our childhood population has yielded only slightly (Chart 1). Today accidents are the greatest single cause of death among children between the ages of 1 and 15 years. In fact, they kill almost as many as all other causes of death combined (Chart 2).

During 1952, 180 Kansas children 14 years of age and under were killed in accidents of all types. Seventy-seven of these occurred in the home, 59 in motor vehicles, and the balance in a variety of other places.

In reviewing the major classifications of fatal home accidents among children 0 to 14 years (Chart 3), it is quite apparent that in the pre-school years most of them occur in or about the home and, as we would expect, as the child grows older, motor vehicle and public accidents become more common. Note the various types of fatal accidents in the home with relation to age groups as shown in Table I.

Little information is available on the non-fatal accident problem in Kansas. National estimates indicate, in the case of home accidents, that for every fatal accident we can expect at least 150 non-fatal injuries serious enough to incapacitate the individual for at least 24 hours. Based on national estimates, we can also expect four accidents resulting in a permanent disability for each fatal home accident reported. Thus, we could expect that during 1952, about 11,500 Kansas children less than 15 years of age were so severely injured by accidents in their homes that they were unable to play normally for at least 24 hours, and that 300 of these injuries resulted in a permanent disability. Add to this the serious injuries and disabilities resulting from motor vehicle and other types of accidents, and the number takes on startling proportions.

ACTIVITIES OF MEMBERS

Dr. James B. Weaver, University of Kansas Medical Center, was elected president of a newly formed six-state orthopedic group to serve Arkansas, Colorado, Kansas, Missouri, Nebraska, and Oklahoma. Dr. H. O. Anderson, Wichita, was named secretary-treasurer.

Dr. Franklin D. Murphy, chancellor of the University of Kansas, was principal speaker at a luncheon meeting of the Kansas Council of Women in Topeka on January 27. His subject was "Together We Progress."

Dr. C. F. Taylor, superintendent of the Norton Tuberculosis sanatorium, has joined the staff of the Larned State Hospital as a consultant. He will make weekly visits to the hospital.

Dr. Frederick S. Brenneman, Moundridge, has been granted a two-year leave of absence from the Harvey County Medical Society to work overseas with the Mennonite mission.

Dr. Robert A. Jordan, who has been practicing in Lawrence, has joined the staff of the University of Kansas Medical Center as an assistant professor in the Department of Internal Medicine.

Dr. L. S. Nelson, Salina, addressed a meeting of licensed practical nurses in District Five at a meeting held in Salina recently.

Dr. M. J. Cox, director of the Ford County Tumor Clinic, addressed the Parent-Teacher Association of Ashland on January 25. He spoke on the subject of cancer and showed two films, "The Doctor Speaks" and "The Warning Shadow."

Dr. Spencer Fast, Atchison, has been appointed part time public health director for the city and county of Atchison.

Dr. Ralph E. White, Garnett, addressed the Rotary Club in that city recently on the subject of diets, energy needs, and obesity.

Dr. William J. Reals, Wichita, is the author of a paper, "Protein-Bound Iodine Determinations in a Military Hospital," published in the December issue of *Military Surgeon.*

Dr. Edward D. Greenwood, Topeka, spoke on "Early Adolescent Adjustment" at Roosevelt Junior High School, Topeka, at a February Parent-Teacher Association meeting.

Dr. W. Clarke Wescoe, dean of the University of Kansas School of Medicine, was guest speaker at the annual health education program sponsored by the Woman's Auxiliary to the Wyandotte County Medical Society.

Dr. H. R. Wagenblast has been appointed assistant director for preventable diseases in the city-county health department in Wichita.

Dr. James L. Diacon and Dr. James L. McGovern, Wellington, have announced that they will soon open an office in Argonia.

Dr. Lewis L. Robbins of the Menninger Foundation, Topeka, was a speaker at the banquet program of the Shawnee County Association for Mental Health, Topeka, on February 2.

Dr. Charles A. Campbell, who has been practicing in Bucklin, has announced plans to begin practice soon at the Health Center, Phillipsburg.

Dr. Willard J. Kiser, Wichita, presented a paper, "Surgical Management of Ulcerative Colitis," before a regional meeting of the International College of Surgeons in Oklahoma City on January 23.

Dr. William Nice, who was recently released from the Army after serving two years at Camp Carson, Colorado, has opened an office in Topeka for the practice of internal medicine and pediatrics.

Dr. Philip W. Morgan, Emporia, spoke on "Teamwork Between Physicians and Nurses" before a recent meeting of the Lyon County Licensed Practical Nurses Association.

Dr. Bernard H. Hall, of the Menninger Foundation, addressed the Highland Park Optimist Club, February 4, on the subject of early treatment for the mentally ill.

Dr. Francis T. Collins, president of the Shawnee County Society, addressed the Shawnee County Medical Assistants' Society at a meeting held February 8 in Topeka. He outlined the work of a liaison committee he had recently appointed to bring about cooperation of the two groups in mutual projects.

Dr. Noble E. Melencamp, Dodge City, was elected "Man of 1954" by the Sigma Phi Epsilon fraternity at Kansas University. The title is awarded annually to the father of one member of the fraternity.

Dr. E. Burke Scagnelli, Dodge City, gave a talk on socialized medicine to the Knights of Columbus at a meeting held in Jetmore, January 31.

Dr. V. J. Vaughn, who has practiced in Axtell for five years, has announced that he will close his office to begin a residency in anesthesiology at Iowa University on April 1.

Dr. Harold H. Jones, Jr., and Dr. Roscoe Morton have been named part-time directors for the Winfield and Arkansas City offices of the Cowley County Health Department. They will serve until a full time director is named.

Capt. Donald W. Selzer, formerly of Manhattan, is now stationed at Fort Riley. He expects to be released from service in April and will begin a surgical residency at the Mayo Clinic in the fall.

Dr. Almonta D. Hays, Cherokee, has found it necessary to discontinue practice because of illness. Dr. M. F. Stock, Weir, will spend two afternoons each week in Cherokee to care for patients of Dr. Hays.

A tribute to Dr. and Mrs. Claire Hays, Cedar Vale, was published as an editorial in the *Cedar Vale Messenger* on February 4. They were credited with carrying forward the plan for a hospital in the city, and the new institution was opened on February 7.

Dr. Orville S. Walters, Topeka, spoke on "A Doctor Thinks About Religion" before a young people's group at the Seward Avenue Baptist Church in Topeka on February 21.

Dr. Daniel W. Melton, Preston, was honored by the community at a covered dish dinner held at the Preston High School on February 17. He has practiced there for 47 years.

Dr. Mary T. Glassen, Phillipsburg, was guest speaker at a meeting of the City Teachers' Association at Concordia on February 16.

Three Independence physicians, Dr. Porter M. Clark, Dr. Gerald C. Bates, and Dr. Harold O. Bullock formed a panel for the first free community medical forum held in Independence under the sponsorship of the Rotary Club there. Dr. Clark spoke on "Your Heart and You," and the others answered medical questions from the floor.

Cost of Medical Education

Medical college training in America costs approximately $9,200 today, according to a survey made recently for the *Journal of Medical Education*. The figures were based on questionnaires sent to students at 26 medical schools throughout the country.

The $9,200 does not cover the cost of premedical training, internship, and residency training, or the cost to the young physician of setting himself up in practice.

One-third of the 6,251 students who answered expect to be in debt when they graduate from medical school, and the amount of debt averages approximately $3,500. About half of the students are single and live away from home. Most of the money for education for the single student comes from his parents, while the married student is supported largely by the earnings of his wife. Income of all students varies widely, with an average of $2,450.

DEATH NOTICES

James Albert Settle, M.D.

Dr. J. A. Settle, 84, an honorary member of the Crawford County Society who had been living in Hutchinson since his retirement in 1949, died there on January 23. He received his education at the University of Tennessee and the University Medical College of Kansas City, winning his medical degree in 1893. His 56 years of practice were spent in the southeastern part of Kansas, 25 years in Walnut. During World War I, he was a captain in the Army Medical Corps, serving at Fort Riley. He had served as secretary and as president of his county society during his years of active practice.

Amendments to Constitution and By-Laws

In accordance with provisions of the constitution of the Kansas Medical Society, the Committee on Constitution and Rules now presents all amendments which have been suggested during the past year. These amendments will be discussed and voted upon at the meetings of the House of Delegates on May 4 and 6, 1954, in Topeka.

Numbers 1 and 2 relate to one subject:

1. Resolved: That the Constitution, Article VI, Section 1, be amended after the word "Society" by inserting a comma and the words "Chairman of the Editorial Board."

ARTICLE VI—COUNCIL

Section 1. The Council shall consist of one Councilor from each councilor district, and in addition the officers of the Society, CHAIRMAN OF THE EDITORIAL BOARD and the delegates to the American Medical Association as ex-officio members.

2. Resolved: That the Constitution, Article VII, Section 1, be amended after the word "councilors," by inserting the words "Chairman of the Editorial Board."

ARTICLE VII—HOUSE OF DELEGATES

Section 1. The House of Delegates shall be the primary legislative and governing body of this Society, and shall consist of the duly elected delegates, and the officers, councilors, CHAIRMAN OF THE EDITORIAL BOARD and past presidents of this Society.

COMMENT: Amendments No. 1 and No. 2 were read at the 1953 meeting and should be voted upon at the 1954 meeting. Their purpose, as explained last year, is to make the Chairman of the Editorial Board an official member of the Council and of the House of Delegates.

The committee recommends that resolutions 1 and 2 be adopted.

Numbers 3, 4, and 5 relate to one subject:

3. Resolved: That the Constitution, Article IV, Section 2, be amended by deleting the words "a second vice-president."

ARTICLE IV—COMPOSITION OF THIS SOCIETY

Section 2. The officers of this Society shall be a president, a president-elect, a first vice-president, [a second vice-president,] a secretary and a treasurer. All officers shall be elected by the House of Delegates of this Society for terms of office as are hereinafter provided.

4. Resolved: That the By-Laws, Chapter VI, Section 1, as amended in 1953, be further amended by deleting the words "First Vice-President," and by deleting the word "Two" and substituting therefore the word "THREE," and by deleting the word "Second" in the next line and substituting therefore the word "FIRST."

CHAPTER VI—ELECTION OF OFFICERS

Section 1. . . . the Committee shall meet not later than 90 days prior to the next annual session and present for publication in the Journal a list of candidates for each elective office, consisting of one or more candidates for the offices of President-Elect, [First Vice-President,] Secretary, Treasurer, and Delegate-Elect to the American Medical Association, and [two] THREE or more candidates for the offices of [Second] FIRST Vice-President and Alternate Delegate to the American Medical Association. . . .

The remainder of this section is not applicable to this amendment so is not reprinted here.

5. Resolved: That the By-Laws, Chapter VII, Section 4, be deleted and that the following sections be appropriately renumbered.

CHAPTER VII—DUTIES OF OFFICERS

[Section 4. The second vice-president shall also assist the president in the performance of his duties and in the event of the death, resignation, or removal of the first vice-president, shall succeed to that office for the remainder of the term.]

COMMENT: The Shawnee County delegation presented two resolutions at the 1953 session dealing with the officers of the Society and their election. Deleting the "Whereases" they were as follows:

(1). *Resolved* that the office of Second Vice-President be abolished, and *Resolved* that the vacancy on the Council, by abolishing the second vice-president, be filled by the Editor of the Journal.

(2). *Resolved* that a minimum of three names be offered as candidates for the office of First vice-president with a provision to permit nominations from the floor.

The first of these would require a constitutional amendment, and, being presented at one meeting, should be voted upon at the succeeding session. It would also require By-Laws changes. Since portions of these Shawnee County amendments are already incorporated in Constitutional amendments No. 1 and No. 2, we consider that amendments 3, 4 and 5,

Clinical Results* with Banthine® Bromide

(Brand of Methantheline Bromide)

22 Published Reports Covering Treatment of 1443 Peptic Ulcer Patients with Banthine

Comprising the reports published in the literature to date which give specific facts and figures of the results of treatment

AUTHORS	No. of Patients	Chronic, Resistant to Other Therapy	TYPES OF ULCERS				RELIEF OF SYMPTOMS (Chiefly Pain)				Surgery or Complications[1]	Side Effects Requiring Discontinuance of Drug[2]	EVIDENCE OF HEALING			
			Duodenal	Jejunal	Stomal	Gastric	Good	Fair	Poor	No Report			Complete	Moderate	None	No Report
Grimson, Lyons, Reeves	100	100	93	7			80	11	4		5		47		19	29
Friedman	15	15	14			1	5		4	6[3]			2			13
Bechgaard, Nielsen, Bang, Gruelund, Tobiassen	26	26	21			5	16	4	6				8	6	12	
McHardy, Browne, Edwards, Marek, Ward	162		162				136	12	11		3	1	14	9	7	129
Segal, Friedman, Watson	34	34	34[4]				14	13			7	2	5		8	14
Brown, Collins	117	99	117				97	7	8		5	8	55	9	8	40
Asher	77		65		7	5	52	9	16			16		9	21	47
Rodriguez de la Vega, Reyes Diaz	5	4	5				4		1					3	2	
Winkelstein	116	116	102	8		6	102		14				53		18	45
Hall, Hornisher, Weeks	18	18	18				11		1	6[5]			18			
Maier, Meili	38	38	24			14[6]	27	7	4[7]				10	2	5	21
Meyer, Jarman	25	18	25				21		4							25
Polh, Fromm	37	37	37				33	3	1				33	3	1	
Plummer, Burke, Williams	41	41	41				36		5				38		3	
McDonough, O'Neil	104	100	104				63	10	31			11	4		11	89
Broders	60	60	58		1	1	35	19	6				10	1	49[8]	
Legerton, Texter, Ruffin	11		11				11									11
Holoubek, Holoubek, Langford	76	69	76				35	27	10		4	10	26		10	36
Dgborn	42		39	2		1	42[9]									42
Shaiken	48	48	48				33	10	3		2		33	10	3	
Johnston	145	145	145				143		2			2	143		2	
Rossett, Knox, Slephenson	146		141			5	146					4[10]	53			93
TOTALS	*1443*	*968*	*1380*	*17*	*8*	*38*	*1142*	*132*	*131*	*12*	*26*	*54*	*552*	*52*	*179*	*634*
PERCENTAGES		*67.8*	*95.6*	*1.2*	*0.6*	*2.6*	*81.3*	*9.4*	*9.3*			*3.7*	*70.5*	*6.6*	*22.9*	

1. Not included in tabulations.
2. Included in "Relief of Symptoms" as "Poor" and in "Evidence of Healing" as "None."
3. Four had no symptoms when Banthine therapy was begun.
4. Of which seven were penetrative lesions and five partially obstructive.
5. No symptoms were present in four.
6. Two with symptoms only; no demonstrable ulcer.
7. Three were psychopathic patients and one had a ventricular ulcer of the lesser curvature.
8. Roentgen findings after treatment period of two weeks; forty-seven had duodenal deformity.
9. All returned to work within a week.
10. In these four, after relief of symptoms, Banthine was discontinued because of urinary retention.

During the past three years, more than 250 references to Banthine therapy in peptic ulcer and other parasympathotonic conditions have appeared in medical literature. Of these reports, 22 have presented specific facts and figures on the results of treatment in a total of 1,443 peptic ulcer patients, 67.8 per cent of whom were reported as chronic or resistant to other therapy. These results are tabulated above and show:

"Good" relief of symptoms was obtained in 81.3 per cent of the 1,405 patients on whom reports were available.

"Complete" evidence of healing was obtained in 70.5 per cent of the 783 patients on whom reports were available.

In all but 9.3 per cent, relief of pain was "good" or "fair." In all but 22.9 per cent, evidence of healing was "complete" or "moderate."

During treatment, 26 patients required surgery or developed complications other than ulcer which required discontinuance of the drug before results could be evaluated.

Of the remaining 1,417 patients, only 3.7 per cent experienced side effects sufficiently annoying to require discontinuance of the drug.

ACCEPTED COUNCIL ON PHARMACY AND CHEMISTRY AMERICAN MEDICAL ASSOCIATION

*Volume containing complete references, with abstracts of 39 additional reports, will be furnished on request by

G. D. Searle & Co.
P. O. Box 5110, Chicago 80, Illinois

if passed, will make the wishes of the delegates from Shawnee prevail.

The Committee recommends that the office of second vice-president NOT be abolished, but favors the requirement that three or more candidates be nominated for the offices of second vice-president and Delegate-Elect to the American Medical Association. Therefore, the committee recommends that amendments 3 and 5 NOT pass and that amendment 4 be altered to include only the substitution of the word "three" for the word "two."

Numbers 6 and 7 relate to one subject:

6. Resolved: That the By-Laws, Chapter XII, Section 7 be amended by inserting a period after the word "Council" and deleting the remainder of that section.

CHAPTER XII—COMPONENT SOCIETIES

Section 7. Any physician who may feel aggrieved by the action of the component society of his county in refusing him membership, or in suspending or expelling him, shall have the right to appeal to the Council. [whose decision shall be final as to whether or not the appellant shall hold membership in this Society.]

COMMENT: Present wording of this section would appear to negate the time-honored right of each component society to be the absolute judge of its membership. The above deletion would still reserve the right of appeal to a censored or rejected member and would allow the Council to make recommendations to the component society.

7. Resolved: That the By-Laws, Chapter XII, Section 8 be amended by inserting after the word "compromise" the following: "Shall be the responsibility or duty of the Councilor of that district, and . . ."

CHAPTER XII—COMPONENT SOCIETIES

Section 8. In hearings of appeals, the Council may admit oral or written evidence as in its judgment will best and most fairly present the facts, but in every case of appeal efforts for conciliation and compromise SHALL BE THE RESPONSIBILITY OR DUTY OF THE COUNCILOR OF THAT DISTRICT, AND shall precede all hearings.

COMMENT: This would emphasize the fact that efforts for conciliation or compromise should be made through the efforts of the councilor of the affected district.

The committee recommends the passage of amendments 6 and 7.

---

8. Resolved: That the By-Laws, Chapter XI, Section 7 be amended by inserting after the words "Executive Committee" the phrase, "who shall determine that the anticipated expenses of the meeting are within the budget."

CHAPTER XI—COMMITTEES

Section 7. The Committee on Arrangements . . . shall prepare and arrange for diversified scientific programs subject to the approval of the Executive Committee WHO SHALL DETERMINE THAT THE ANTICIPATED EXPENSES OF THE MEETING ARE WITHIN THE BUDGET and shall have general charge of all the annual session arrangements. . . .

The remainder of the section does not apply to this resolution.

COMMENT: This amendment was suggested in order that under no circumstances can the arrangements committee plan for and carry on a program for a meeting whose cost will be in excess of available funds of the Society.

The committee approves the adoption of this resolution.

Numbers 9 and 10 relate to one subject:

9. Resolved: That the By-Laws, Chapter IX, Section 1 be amended by deleting the entire section after the word "defense" in line 11 and substituting the words "To assist such member in planning for defense and IF REQUESTED by the defendant and deemed advisable by the Defense Board procure the services of an attorney approved by The Kansas Medical Society, provided the entire cost of such services shall be paid by the defendant. The Society shall not be obligated to pay ANY fees for legal services, nor any costs or judgments arising out of any action involving the professional services of any member."

CHAPTER IX—DEFENSE BOARD

Section 1. Members of The Kansas Medical Society are urged to carry individual professional insurance with commercial companies as these usually include possible indemnity in their policies, but additional assistance of the Society Defense Counsel may be provided if necessary or desired. It shall be the duties of the members of the Defense Board, severally or collectively, to investigate all claims of malpractice made against members desiring Society assistance; to assist in conducting the defense; [and to pay the Society defense attorney his fee and expenses from funds of this Society within their control, but they shall not pay or obligate this Society to pay any court costs or judgments rendered against any member in the final determination of any case, nor shall they be obligated without affirmative action, to pay

in arthritis
and allied disorders

BUTAZOLIDIN

(brand of phenylbutazone)

ACCEPTED COUNCIL ON PHARMACY AND CHEMISTRY AMERICAN MEDICAL ASSOCIATION

potent, non-hormonal antiarthritic agent

Its therapeutic effectiveness substantiated by more than fifty published reports, BUTAZOLIDIN has recently received the Seal of Acceptance of the Council on Pharmacy and Chemistry of the American Medical Association.

In the treatment of arthritis BUTAZOLIDIN produces prompt relief of pain. In many instances relief of pain is accompanied by diminution of swelling, resolution of inflammation and increased freedom and range of motion of the affected joints.

BUTAZOLIDIN is indicated in:

Gouty Arthritis — Rheumatoid Arthritis
Psoriatic Arthritis — Rheumatoid Spondylitis

Painful Shoulder (including peritendinitis, capsulitis, bursitis, and acute arthritis)

Since BUTAZOLIDIN is a potent agent, patients for therapy should be selected with care; dosage should be judiciously controlled; and the patient should be regularly observed so that treatment may be discontinued at the first sign of toxic reaction.

Physicians unfamiliar with the use of BUTAZOLIDIN are urged to send for complete descriptive literature before employing it.

BUTAZOLIDIN® (brand of phenylbutazone), coated tablets of 100 mg.

Geigy

GEIGY PHARMACEUTICALS
Division of Geigy Chemical Corporation
220 Church Street, New York 13, N. Y.
In Canada: Geigy Pharmaceuticals, Montreal

360

the fees of any local attorney employed by the member. The Defense Board shall be empowered to contract with agents or attorneys as it deems necessary, insofar as the amounts to be expended thereby are approved, or do not exceed the amount of the annual budget for defense purposes.] TO ASSIST SUCH MEMBER IN PLANNING FOR DEFENSE AND IF REQUESTED BY THE DEFENDANT AND DEEMED ADVISABLE BY THE DEFENSE BOARD PROCURE THE SERVICES OF AN ATTORNEY APPROVED BY THE KANSAS MEDICAL SOCIETY, PROVIDED THE ENTIRE COST OF SUCH SERVICES SHALL BE PAID BY THE DEFENDANT. THE SOCIETY SHALL NOT BE OBLIGATED TO PAY ANY FEES FOR LEGAL SERVICES, NOR ANY COSTS OR JUDGMENTS ARISING OUT OF ANY ACTION INVOLVING THE PROFESSIONAL SERVICES OF ANY MEMBER.

10. Resolved: That the By-Laws, Chapter IX, Section 6 be amended by deleting the first two sentences and substituting therefore "Section 6. Disbursements for defense to include reimbursement for any actual expenses incident to travel, by members of the Defense Board, required in the performance of official duties, and a per diem sum fixed by the Council for the days actually spent in such official duties may be allowed."

CHAPTER IX—DEFENSE BOARD

Section 6. [Disbursements for defense shall be annually estimated in advance by the Defense Board as based upon average expenses of the preceding two years. The computed amount shall then be included in the annual budget of the Executive Secretary for presentation to and approval by the House of Delegates.] DISBURSEMENTS, FOR DEFENSE TO INCLUDE REIMBURSEMENT FOR ANY ACTUAL EXPENSES INCIDENT TO TRAVEL, BY MEMBERS OF THE DEFENSE BOARD, REQUIRED IN THE PERFORMANCE OF OFFICIAL DUTIES, AND A PER DIEM SUM FIXED BY THE COUNCIL FOR THE DAYS ACTUALLY SPENT IN SUCH OFFICIAL DUTIES MAY BE ALLOWED.

The remainder of the section will remain but has no bearing upon the amendment so is not reprinted here.

COMMENT: These two amendments were prepared at the request of the Council. Reasons for this change from the original plan of providing services of the attorney for the Society when requested can best be explained by them.

The committee recommends adoption of these two amendments.

---

11. Resolved: That the By-Laws, Chapter II, Section 1 be amended by striking out the words "Twenty-five" and substituting the word "Fifty."

CHAPTER II—ASSESSMENTS

Section 1. The amount of the annual assessment for The Kansas Medical Society only shall be not more than [twenty-five] FIFTY dollars per member, the exact amount to be determined by the Council after consideration of the annual budget for the ensuing year and to be announced to the various component societies not less than three months before the beginning of each fiscal year. . . .

The remainder of this section has no bearing on this amendment.

COMMENT: Increased activities and increased costs generally have made expenses exceed the income for the past few years. Rather than delve into reserve funds established years ago, this increase of dues is recommended. While fifty dollars is set as a maximum, it is presumed that a lesser sum may be set by the Council annually.

The committee recommends adoption of this resolution.

---

12. Resolved: That the By-Laws, Chapter I, Section 4 be amended by deleting the last sentence.

CHAPTER I—MEMBERSHIP

Section 4. . . . [Honorary members having qualifications required by the Constitution of the American Medical Association may be recommended for Affiliate Fellowship in the American Medical Association by vote of the House of Delegates.]

The preceding portion of this section has no bearing on this amendment.

COMMENT: Since fellowship classifications within the American Medical Association have been abolished, this sentence has been rendered inoperative.

The committee recommends adoption of this resolution.

Numbers 13 and 14 pertain to the same subject:

13. Resolved: That the By-Laws, Chapter XI, Section 1a, be amended by adding a new section 37 "Committee on General Practice Award."

14. Resolved: That the By-Laws, Chapter XI be amended by adding a section.

CHAPTER XI—COMMITTEES

SECTION 37. THE COMMITTEE ON GENERAL PRACTICE AWARD SHALL CONSIST OF FIVE OR MORE MEMBERS OF WHICH ONE SHALL BE A COUNCILOR AND ONE THE PRESIDENT OF THE KANSAS ACADEMY OF



Thank you doctor
for telling mother about...



The Best Tasting Aspirin you can prescribe

The Flavor Remains Stable down to the last tablet

Bottle of 24 tablets 15¢ (2½ grs. each)

We will be pleased to send samples on request

THE BAYER COMPANY DIVISION of Sterling Drug Inc., 1450 Broadway, New York 18, N. Y.

GENERAL PRACTICE OR A MEMBER OF THAT ACADEMY DESIGNATED BY HIM. TWO OR MORE MEMBERS SHALL HAVE SERVED ON THE RETIRING COMMITTEE. THE DUTIES OF THE COMMITTEE SHALL BE TO DETERMINE AN INDIVIDUAL MEMBER AND SUBMIT ADEQUATE MATERIAL AND INFORMATION IN SUPPORT OF ANY KANSAS MEDICAL SOCIETY MEMBER DESIRED TO BE SUBMITTED TO THE AMERICAN MEDICAL ASSOCIATION COMMITTEE AS A CANDIDATE FOR THE ANNUAL GENERAL PRACTITIONER'S AWARD.

COMMENT: This will establish a permanent mechanism whereby this Society may nominate, if it wishes to do so, a candidate for the American Medical Association award to the outstanding general practitioner of the year.

The committee recommends adoption of these resolutions.

---

15. Resolved: That the By-Laws, Chapter VIII, Section 12, be amended by striking the word "Eleven" and substituting "a majority of the."

CHAPTER VIII—THE COUNCIL

Section 12. [Eleven] A MAJORITY OF THE members of the Council shall constitute a quorum.

COMMENT: This appears advisable to care for this question regardless of the number on the Council.

The committee recommends this be passed.

Numbers 16, 17, 18, and 19 refer to one subject:

COMMENT: Revision of the Council districts in an effort to produce a more equitable distribution of members within the several districts has been a project with which your committee has worked during this past year. The following four plans appear feasible.

The committee recommends that the House of Delegates select its preference from these four alternatives:

Number 16 retains 12 council districts.

Number 17 creates 15 council districts.

Number 18 creates 14 council districts.

Number 19 creates 17 council districts.

16. Resolved: That the By-Laws, Chapter VIII, Section 13, enumerating the counties comprising the present Councilor Districts remain as at present except that Leavenworth County be removed from District 2 and placed into District 1.

CHAPTER VIII—THE COUNCIL

Section 13. The councilor districts shall be comprised of the following counties: First District: Marshall, Nemaha, Brown, Doniphan, Pottawatomie, Jackson, Atchison, [and] Jefferson AND LEAVENWORTH Counties. Second District: [Leavenworth,] Wyandotte, Douglas, Johnson, Franklin, Miami, Anderson and Linn Counties.

The remainder of this long section is not quoted because it remains as at present.

COMMENT: This will occasion no change in Councilor elections.

17. Resolved: That the By-Laws, Chapter VIII, Section 13 enumerating the present Councilor Districts shall retain their present boundaries except that Leavenworth County be placed into District 1 and that three new districts shall be formed whereby Wyandotte County shall be removed from District 2 and become District 13, Shawnee County shall be removed from District 4 and become District 14 and Sedgwick County shall be removed from District 6 and become District 15.

COMMENT: This will alter counties as currently listed in this section but because of its length is not reprinted here. Such action will not conflict with other sections of the Constitution and By-Laws and will require the election of five councilors each year for the future.

If this amendment is voted the following, in addition to the regular Councilor elections, will become necessary during this annual session.

a. District 4 (without Shawnee County) would elect a Councilor for a term expiring in 1957.

b. District 6 (without Sedgwick County) would elect a Councilor for a term expiring in 1956.

c. District 13 (Wyandotte County) would elect a Councilor for a term expiring in 1955.

18. Resolved: That the By-Laws, Chapter VIII, Section 13 be deleted after the word "counties" and the following be inserted:

District 1—Atchison, Brown, Doniphan, Jackson, Jefferson, Leavenworth and Nemaha.

District 2—Anderson, Douglas, Franklin, Johnson, Linn, Miami and Wyandotte.

District 3—Allen, Bourbon, Cherokee, Crawford, Labette, Montgomery, Neosho, Wilson and Woodson.

District 4—Clay, Geary, Marshall, Pottawatomie, Riley and Washington.

District 5—Chase, Coffey, Lyon, Morris, Osage, Shawnee and Wabaunsee.

District 6—Butler, Chautauqua, Cowley, Elk and Greenwood.

District 7—Cloud, Dickinson, Ellsworth, Jewell, Lincoln, Mitchell, Ottawa, Republic and Saline.

District 8—Harvey, McPherson, Marion, Reno and Rice.

District 9—Barber, Harper, Kingman, Sedgwick, and Sumner.

℞

Neo-Synephrine®

hydrochloride

Running noses, sneezing, watery eyes, clogged-up nasal passages quickly yield to administration of Neo-Synephrine hydrochloride — a nasal decongestant of proved clinical value. Ciliary activity is nearly untouched, sting and congestive rebound are practically absent, and effectiveness is undiminished on repeated use throughout the cold season.



Neo-Synephrine HCl

- 0.25% Solution
- 0.25% Spray (unbreakable plastic squeeze bottle)
- 0.25% Solution (Aromatic)
- 0.5% Solution
- 1% Solution
- 0.25% Emulsion
- 0.5% Jelly

Winthrop-Stearns INC.
New York 18, N. Y. • Windsor, Ont.

Neo-Synephrine, trademark reg. U.S. Pat. Off.,
brand of phenylephrine

District 10—Ellis, Graham, Osborne, Phillips, Rooks, Russell, Smith and Trego.

District 11—Barton, Hodgeman, Ness, Pawnee, Rush and Stafford.

District 12—Clark, Comanche, Edwards, Ford, Gray, Kiowa, Meade, Pratt and Seward.

District 13—Cheyenne, Decatur, Gove, Logan, Norton, Rawlins, Sheridan, Sherman, Thomas and Wallace.

District 14—Finney, Grant, Greeley, Hamilton, Haskell, Kearny, Lane, Morton, Scott, Stanton, Stevens and Wichita.

COMMENT: If this resolution is adopted, it is recommended that each present councilor retain his position until the expiration of his present term and that new councilors be elected in the newly formed districts, their terms to be staggered as indicated to insure proper rotation.

One conflict is created because two present councilors reside in the proposed District 12. It is recommended that both continue in their office for one year, at which time Doctor Klein will be ineligible for re-election because of having completed two successive terms. Then in 1956, when Doctor Black's second term expires, District 12 will elect a new councilor for a three-year term.

District	*Councilor*	*Term Expires*
1	W. L. Anderson, M.D., Atchison	1954
2	H. P. Jones, M.D., Lawrence	1954
3	Charles E. Vestle, M.D., Humboldt	1955
4	S. A. Anderson, M.D., Clay Center	1954
5	F. C. Taggart, M.D., Topeka	1956
6	NEW	1957
7	A. E. Rueb, M.D., Salina	1954
8	H. M. Glover, M.D., Newton	1956
9	N. L. Francis, M.D., Wichita	1955
10	L. W. Reynolds, M.D., Hays	1955
11	NEW	1955
12	C. V. Black, M.D., Pratt	1956
	R. G. Klein, M.D., Dodge City	1955
13	J. L. Jenson, M.D., Colby	1956
14	NEW	1956

19. Resolved: That the By-Laws, Chapter VIII, Section 13 be deleted after the word "counties" and the following be inserted:

District 1—Atchison, Brown, Doniphan, Jackson, Jefferson, Leavenworth and Nemaha.

District 2—Wyandotte County.

District 3—Anderson, Douglas, Franklin, Johnson, Linn and Miami.

District 4—Allen, Bourbon, Cherokee, Crawford, Labette, Montgomery, Neosho, Wilson and Woodson.

District 5—Clay, Geary, Marshall, Pottawatomie, Riley and Washington.

District 6—Shawnee County.

District 7—Chase, Coffey, Lyon, Morris, Osage and Wabaunsee.

District 8—Butler, Chautauqua, Cowley, Elk and Greenwood.

District 9—Cloud, Dickinson, Ellsworth, Jewell, Lincoln, Mitchell, Ottawa, Republic and Saline.

District 10—Harvey, McPherson, Marion, Reno and Rice.

District 11—Sedgwick County.

District 12—Barber, Harper, Kingman, Pratt and Sumner.

District 13—Ellis, Graham, Osborne, Phillips, Rooks, Russell, Smith and Trego.

District 14—Barton, Hodgeman, Ness, Pawnee, Rush and Stafford.

District 15—Clark, Comanche, Edwards, Ford, Gray, Kiowa, Meade and Seward.

District 16—Cheyenne, Decatur, Gove, Logan, Norton, Rawlins, Sheridan, Sherman, Thomas and Wallace.

District 17—Finney, Grant, Greeley, Hamilton, Haskell, Kearny, Lane, Morton, Scott, Stanton, Stevens and Wichita.

COMMENT: This varies from resolution No. 10 in the separation of the three large counties and in changing Pratt County.

If this resolution is adopted, the following situation would apply regarding Councilor elections at this session. No conflicts are created.

District	*Councilor*	*Term Expires*
1	W. L. Anderson, M.D., Atchison	1954
2	NEW	1955
3	H. P. Jones, M.D., Lawrence	1954
4	C. E. Vestle, M.D., Humboldt	1955
5	S. A. Anderson, M.D., Clay Center	1954
6	F. C. Taggart, M.D., Topeka	1956
7	NEW	1956
8	NEW	1957
9	A. E. Rueb, M.D., Salina	1954
10	H. M. Glover, M.D., Newton	1956
11	N. L. Francis, M.D., Wichita	1955
12	C. V. Black, M.D., Pratt	1956
13	L. W. Reynolds, M.D., Hays	1955
14	NEW	1955
15	R. G. Klein, M.D., Dodge City	1955
16	J. L. Jenson, M.D., Colby	1956
17	NEW	1957

New Parke Davis Advertisement

A new Parke Davis and Company advertisement, circulated widely through lay publications, gives timely advice on "How to Select a Family Doctor." The emphasis of the copy is placed on selecting a doctor before a time of crisis. Three suggestions are given: "(1) Ask your county medical society for a list of qualified physicians in your community; (2) Or, ask a local hospital or your pharmacist for the names of three or more doctors; (3) Visit several doctors, if you wish, before you make your choice. Mutual confidence is an important factor in this relationship."

K.M.S. annual meeting, May 2-6, 1954, Topeka.

tested

broad-spectrum therapy

Terramycin®

BRAND OF OXYTETRACYCLINE

established

an agent of choice

in the treatment of a wide range of infections due to gram-positive and gram-negative bacteria, spirochetes, rickettsiae, certain large viruses and protozoa.

clinical advantages known

rapid absorption

wide distribution

prompt response

excellent toleration

Within an hour after oral administration in fasting or non-fasting state, effective serum concentrations of Terramycin may be attained.[1] It is widely distributed in body fluids, organs and tissues and diffuses readily through the placental membrane.[2,3] Immediate evidence of Terramycin's efficacy is often obtained by the rapid return of temperature to normal.[4] Widely used among patients of all ages, this tested broad-spectrum antibiotic is well tolerated.[5,6]

1. Sayer, R. J., et al.: Am. J. M. Sc. *221*:256 (Mar.) 1951.
2. Welch, H.: Ann. New York Acad. Sc. *53*:253 (Sept.) 1950.
3. Werner, C. A., et al.: Proc. Soc. Exper. Biol. & Med. *74*:261 (June) 1950.
4. Wolman, B., et al.: Brit. M. J. *1*:419 (Feb. 23) 1952.
5. Potterfield, T. G., et al.: J. Philadelphia Gen. Hosp. *2*:6 (Jan.) 1951.
6. King, E. Q., et al.: J. A. M. A. *143*:1 (May 6) 1950.

Available in convenient oral, parenteral and ophthalmic preparations.

Pfizer

PFIZER LABORATORIES, *Brooklyn 6, N. Y.*
Division, Chas. Pfizer & Co., Inc.

The Pathogenesis of Hypochromic Microcytic Anemia

David L. Zacharias, M.D.

Kansas City, Missouri

HISTORICAL

This history of hypochromic microcytic anemia, as such, extends back some 75 years, when Hayem first observed what we would now designate as a lowered color index in the anemia associated with chlorosis. In 1883 Laache showed that the proportion of hemoglobin to the number of red cells was of some importance in differentiating the anemias, and in 1885 Osler recognized that after severe hemorrhage the development of hemoglobin did not keep pace with the red cell formation.

Although Sydenham had found in 1661 that chlorosis was benefited by treatment with iron, no rational explanation was offered until 1746, when Menghinis discovered that this element was a characteristic part of the blood. Fodish demonstrated in 1832 that the iron content in the blood of chlorotics was greatly decreased, and in the same year Blaud announced the formula and dosage of an iron preparation which was effective in the treatment of chlorosis. There were some, however, who questioned the value of iron in the treatment of anemia even up to and past the turn of the present century, but since 1926, when Minot and Murphy demonstrated the value of liver in pernicious anemia, the value of iron as a specific remedy for certain anemias seems to have been permanently established.[1, 2, 3]

ETIOLOGY

That a hypochromic microcytic anemia can be produced by several deficiency states has been shown experimentally in the laboratory animal, but fortunately not all of these have been demonstrated in man. Such anemias have been observed in swine fed a diet lacking in pyrodoxine or pantothenic acid[4] and in rats kept deficient in copper,[5] and in 1935 Underwood and Filmer demonstrated that a deficiency of cobalt in grazing areas was ofttimes responsible for such a condition in cattle and sheep. Since deficiencies of pyrodoxine, pantothenic acid, or cobalt have never been demonstrated in man, and copper deficiency very rarely, the bulk of the hypochromic anemia seen clinically must be due to some other cause, which is, of course, a deficit in the amount of iron available for adequate erythrocyte production.

The anemia associated with chronic infection is most often of a normocytic normochromic type, but the cells may become pale and small after the condition has been present for some time.[6] Likewise, the anemia of sprue, while classically a macrocytic hyperchromic type, may in the later stages come to be hypochromic and microcytic. In all probability, the changed morphology of the red cells in the later stages of both of these states is due to a lack of iron being presented to the bone marrow for red cell production with a consequent over-shadowing of the original condition by iron deficiency.

The importance of iron deficiency can be demonstrated by noting that it is the most common clinically manifest deficiency state occurring in some institutions. It occurs most often in women during the child bearing age and at the menopause, in infants of either sex between the ages of 8 and 27 months,[7] and in adolescent girls with the combined requirments of rapid growth and the menses.[1] A hypochromic microcytic or iron deficiency anemia seldom occurs in the adult male without chronic blood loss, although it is sometimes seen in the adolescent. Even here it is far from common.

In order to explain the mechanism and typical group occurrence of this anemia, it will be necessary to review briefly the metabolism of iron. According to all available evidence, and volumes have been published during the past few decades, iron is one of the few substances which the human body has little ability to excrete through the ordinary channels. The amount lost by way of the intestinal tract has been estimated as approximately 0.3 to 0.5 milligram daily, and measurable amounts are lost through perspiration, desquamated epithelial cells, and from leucocytes discharged from the various body orifices.[8] Urinary loss has been measured in amounts ranging from 0.01 to 0.8 milligram per day, with the smaller figure probably representing the more accurate determination.[9] A small quantity is also used in hair growth, somewhat less than a milligram is lost daily in the milk of the lactating female[1] and approximately 15 to 35 milligrams are lost with each average menstrual flow.[10] Therefore, a relatively small amount of iron, in the neighborhood of ½ to 1 milligram daily, is

This is one of 11 theses, written by fourth year students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group judged to be the best by the faculty at the school.

Thesis written while the author was a fourth year student. Dr. Zacharias is now interning at St. Joseph Hospital, Kansas City, Missouri.

BASIC chemically

The structure of this ***newest*** antibiotic represents a nucleus of modern broad-spectrum antibiotic activity.

BASIC clinically

This ***newest*** broad-spectrum antibiotic has a wide range of action against respiratory, gastrointestinal, soft-tissue, urinary and mixed bacterial infections due to pneumococci, streptococci, staphylococci and other gram-positive and gram-negative organisms.

"Data thus far available would indicate that the use of tetracycline is accompanied by a significantly lower incidence of gastrointestinal symptoms . . ."[2]

This ***newest*** broad-spectrum antibiotic may often be used with good success in patients in whom resistance or sensitivity to other forms of antibiotic therapy has developed.

racyn

brand of TETRACYCLINE hydrochloride

BASIC among broad-spectrum antibiotics

supplied:

TETRACYN TABLETS (sugar coated)
250 mg., 100 mg., 50 mg.



J. B. ROERIG AND COMPANY, Chicago 11, Illinois

all that is necessary to maintain iron balance. Somewhat more is required in the female during periods of rapid growth, pregnancy, or heavy menstruation. The usual yearly requirements of iron for males and females, as determined by Heath and Patek, are listed in the table below.

YEARLY REQUIREMENTS OF IRON FOR MALES AND FEMALES

Age	*Annual Requirements Male in mg.*	*Annual Requirements Female in mg.*
Birth	0.195	0.182
1	0.112	0.112
2	0.080	0.092
3	0.092	0.080
4	0.098	0.087
5	0.079	0.106
6	0.080	0.078
7	0.070	0.067
8	0.072	0.108
9	0.152	0.120
10	0.130	0.163
11	0.137	0.164
12	0.189	0.192
13	0.198	0.145
14	0.314	0.468
15	0.313	0.424
16	0.353	0.498
17	0.183	0.435
18	0.149	0.452
19	0.081	0.365
20	0.071	0.358
21		0.298
22		0.298
23		0.298
24		0.374*
25		0.298

* Iron requirement for pregnancy.

In view of the low requirements necessary to meet physiological needs, an iron deficiency anemia in the adult must be considered as evidence of abnormal loss. The major losses are from hemorrhage and pregnancies. Once the body has attained adult size, it will not become deficient in iron except through one or both of these processes. The deficiency may be accelerated, however, by a poor dietary intake or by poor absorption from the intestinal tract.[8]

In studies of iron metabolism with radioactive isotopes of iron, it has been demonstrated that the ferrous form is absorbed much more readily than the ferric and that the absorption is normally a well-controlled process. Such a mechanism is necessary since the human body is apparently incapable of excreting iron in appreciable amounts, and body levels are ordinarily kept within rather constant limits. In the nondeficient person, only a small portion of the ingested food-iron is absorbed daily, while in the deficient patient the absorptive process is speeded up and is much more efficient. Normal persons may retain only 1 to 10 per cent of a single dose of radioactive iron, while those who are deficient may retain more than 20 per cent.[10] In children, the absorption closely parallels the need of the element, and the same is apparently true during the latter half of pregnancy when the fetal iron stores are being built up.[11]

Since iron is best absorbed in the ferrous state, any reducing substance in the gastrointestinal tract and any acid present are of importance. The acidity of the stomach serves two primary functions. The lowered pH converts colloidal ferric hydroxide normally found in foodstuffs into the molecular ferric ion, and in this acid medium other substances are capable of reducing the ferric ions to the ferrous form. The reducing agents include asorbic acid, cystiene, and the –SH groups of various proteins. Reduction will not occur if the pH is 7 or above.

Iron absorption may be possible in nearly any part of the small bowel, but normally little is taken in except in the duodenal region. Since the reaction here is acid, the ferrous iron formed by reduction in the stomach is not readily oxidized to the ferric form, there is a higher concentration of ferrous ion present, and the absorption appears to be more efficient. As the iron moves on down the intestinal tract, the digestive juices of the pancreas, liver, and intestine tend to raise the pH, and much of the ferrous iron is auto-oxidized again to form ferric hydroxide. With the usual food intake of 2 to 10 milligrams of iron, absorption is probably limited to a very short segment below the pylorus, while with doses of ferrous iron a larger surface may be brought to play.[12] Even then, a maximum of about 15 milligrams is all that can be absorbed from a total dose of 300 milligrams of ferrous iron.

Since the human body appears incapable of excreting iron in any but minimal amounts, the mucosal cells must be capable of limiting absorption by a one-way transfer of iron into themselves. In the normal person there is ordinarily a resistance to the uptake of iron, since absorption can be increased 5 to 20 times in the deficiency state. This limitation is apparently set up within the mucosal cell through the apoferratin-ferratin mechanism.

The present hypothesis is that ferrous iron is taken into the mucosal cell where it is oxidized to the ferric state and incorporated into the protein apoferratin, which is capable of taking up 23 per cent of its own weight of iron and is then designated as ferratin. In order to enter the blood stream, the ferric iron is again split off and reduced to ferrous iron which can penetrate the mucosal cell wall into the vessels. There it is again auto-oxidized to the ferric state and attached to a globulin, siderophilin, for transport. It has been postulated that in the deficiency state the content of apoferratin in the mucosal cells is increased, thereby accounting for the more efficient uptake of iron, and the lesser amount in the average normal cell limits the intake to that required for the needs of the body —the so-called mucosal block.[12]

The absorption of oral doses of radioactive iron is reflected by an increase in the plasma iron in direct proportion to the size of the dose given. This holds true until the dose exceeds that of maximum absorption or until the motility of the gastrointestinal tract



a good "mixer"

for your cough prescriptions

especially valuable when allergic factor

is suspected or present

Chlor-Trimeton syrup q.s. ad

- taste appeals to young and old
- compatible with commonly prescribed medications

Contains CHLOR-*TRIMETON*® Maleate
(brand of chlorprophenpyridamine maleate), 2 mg. per teaspoonful (4 cc.).

Schering

CHLOR-*TRIMETON* SYRUP

SCHERING

is interfered with. The normal range is usually stated to be somewhere between 80 and 180 micrograms per 100 cubic centimeters, but actually there is enough siderophylin present to combine with 300 to 400 micrograms of iron. The plasma level may reach these heights after parenteral iron is given, but it returns to normal within the course of a few hours.

Iron appears to be stored primarily in the liver, spleen, bone marrow, and kidneys, in the form of ferratin, although some probably exists in the form of hemosiderin and monomolecular hydroxides. It also forms an integral part of several enzymes and as such is not available for other uses.[10] Grancik's concept is that the iron available for erythropoiesis exists in two rather different states: the labile iron, composed of recently absorbed iron and that recently liberated from hemoglobin breakdown, and as the less readily available storage iron, composed of somewhat more complex polymers. This is borne out by the fact that the total dose of radioactive iron, given intravenously to deficient patients, appears within 10 days in the red cells. Apparently none is stored for future use nor is there any mixing of the parenteral iron with the already existent stores.[14, 15]

Copper is necessary for the incorporation of iron into protoporphyrin for the formation of the heme portion of hemoglobin. A deficiency may cause an anemia identical with that of iron deficiency. Fever of any origin has been shown to interfere with the utilization of iron, and lead poisoning is also capable of disrupting erythrocyte formation.[16]

The fetus apparently stores iron in the last trimester and may be born with inadequate supplies if the mother is deficient. This is especially true in the case of prematurity or multiple births. The mother's condition is usually not caused by the pregnancy but by factors antedating it, such as bleeding from any cause or repeated pregnancies. That younger siblings tend to develop more severe anemia than their older brothers or sisters has been shown in several families. Contrary to popular belief, a varied diet is not adequate to prevent or relieve anemia in an infant born with inadequate iron storage. Supplemental iron preparations are necessary.[7]

In the hypochromic microcytic anemia of pregnancy, the usual hemoglobin determinations are invalid, due to the rapidly changing plasma volume. The degree of anemia can be evaluated only by a study of the peripheral blood and a determination of the total red cell mass and plasma volume. This anemia is almost invariably present before gestation or very early in pregnancy and appears to be due to factors which were operating before conception occurred. Repeated pregnancies, with iron loss to the fetus and blood loss at the time of delivery, may lead to the development of an iron deficiency anemia, but the fetal uptake during the course of one pregnancy is insufficient to interfere with the maternal hemoglobin level.[17, 18]

The iron deficiency type of anemia sometimes seen in the adolescent female is ordinarily ascribed to the increased requirements which go along with rapid growth and menstruation. A few cases of hypochromic anemia in the adolescent male have been reported, none of which had a history of chronic blood loss but all of which had marked hypochlorhydria or achlorhydria. This suggests a connection between gastric deficiency and anemia.[19]

A hypochromic anemia in the adult can usually be identified with repeated pregnancies or with chronic blood loss. The latter may be due to any number of things, including menorrhagia, bleeding ulcers, bleeding hemorrhoids, abortion or miscarriage, hookworm or other parasites of the gastrointestinal tract, malignancy with bleeding, polyps of the gut, diaphragmatic hernia, or ulcerative colitis. Menopausal menorrhagia is a not uncommon factor in middle aged females.

SYMPTOMATOLOGY

The symptoms of hypochromic anemia are insidious in their onset, and many of the complaints consist of weakness, dyspnea on exertion, easy fatigability, pallor, palpitation, and a feeling of dead tiredness. Rarely, a patient will complain of dysphagia. Pallor of the skin, mucus membranes, and nail beds is in proportion to the reduction of hemoglobin; spoon nails with longitudinal ridging may be present, and an occasional patient may have cheilosis. Hemic murmurs may be present, and in more severe cases there may be edema, anginal symptoms, cardiac enlargement, and electrocardiographic changes. Cheilosis or other oral signs are probably due to a co-existent vitamin deficiency, usually of the B group, and the degree and type of deficiency does not detectably interfere with iron absorption or utilization.[10, 11, 20]

The lower limit of normal hemoglobin levels is somewhat uncertain but is usually considered to be approximately 11.5 grams per cent for the adult white female, 12 grams per cent for the adult white male, and 11 grams per cent for children under the age of 12 years. The values in Negroes may be slightly lower. However, persons with hemoglobin values at or slightly below these levels are not necessarily anemic and may fail to respond to therapy. In the diagnosis of hypochromic anemia, a low hemoglobin is not sufficient; the red cells must also show a mean corpuscular volume of 80 cubic microns or below, and the mean corpuscular hemoglobin concentration must be below 30 per cent. Serum iron determinations are useful in some cases but are not generally available or necessary in the routine case.[10, 11]

50 *and seven*

YEARS TREATING ALCOHOL AND DRUG ADDICTION

In 1897 Doctor B. B. Ralph developed methods of treating alcohol and narcotic addiction that, by the standards of the time, were conspicuous for success.

Twenty-five years ago experience had bettered the methods. Today with the advantages of collateral medicine, treatment is markedly further improved.

The Ralph Sanitarium provides personalized care in a quiet, homelike atmosphere. Dietetics, hydrotherapy and massage speed physical and emotional re-education. Cooperation with referring physicians. Write or phone.

The

RALPH

SANITARIUM

A Unit of the Benjamin Burroughs Ralph Foundation for Medical Research

Telephone VIctor 3624



529 HIGHLAND AVENUE • KANSAS CITY 6, MISSOURI

Ralph Emerson Duncan, M.D., Medical Director.

TREATMENT

Once the diagnosis of iron deficiency anemia is established, the treatment consists simply of the administration of a suitable iron preparation. This should probably consist of a ferrous salt of iron, since it appears that this form is more effective, and could be of either ferrous sulfate, gluconate, chloride, or carbonate. The usual dosage consists of 5 grains of any of these compounds 4 times daily. Larger doses may sometimes cause gastrointestinal discomfort, and massive overdoses have been known to cause acute hemorrhagic gastritis and liver damage.[10, 11]

The use of intravenous saccharated iron oxide has recently received much publicity but appears to be no more effective in the treatment of hypochromic anemia than are the oral preparations. Possible indications for its use include cases with extensive bowel resection, those receiving deep radiation therapy, and perhaps in some cases of severe anemia late in pregnancy when a rapid repletion of the iron stores is desirable.

Toxic reactions to saccharated iron oxide do occasionally occur and may be of a localized or general nature. The former results from extravasation of the solution into the tissues and consists of local pain, perhaps followed by a local thrombophlebitis. Generalized symptoms vary considerably but may include flushing, syncope, headache, substernal distress, shortness of breath, epigastric distress followed by a temperature elevation, hypertension, or tachycardia. Experience indicates that the preparation is safe and relatively non-toxic, but further clinical evaluation is needed. Overdosage may conceivably result in excessive iron deposition with resultant tissue damage.[17, 21, 22]

SUMMARY

Although several factors may be responsible for the production of a hypochromic microcytic anemia, the most important is that of iron deficiency. This is so prominent that some authors consider it as an almost specific etiologic agent. A brief review of the metabolism of iron has been given in an attempt to explain the prevalence and mode of production of this anemia in the different age groups. The symptomatology and treatment have also been mentioned.

BIBLIOGRAPHY

1. Heath, C. W., and Patek, A. J.: The anemia of iron deficiency, Medicine 16:267 (Sept.) 1937.
2. Hahn, P. H.: The metabolism of iron, Medicine 16:249 (Sept.) 1937.
3. Fowler, W. M.: Chlorosis, an obituary, Annals of Medical History, 1936.
4. Wintrobe, Maxwell M.: Physiologic implications of the anemic state, Symposia on Nutrition; Vol. I, Nutritional Anemia, Cincinnati, The Robert Gould Research Foundation, Inc., 1947.
5. Smith, S. E., and Medlincott, M.: The blood picture in iron and copper deficiency, American Journal of Physiology 141, 1944.
6. Cartwright, G. E.; Lauritsen, M. A.; Jones, P. T., and Wintrobe, M. M.: The anemia of infection, Journal of Clinical Investigation 25:1 (Jan.) 1946.
7. Guest, George M.: Hypoferric anemia in infancy, Symposia on Nutrition, Vol. I, Nutritional Anemia, Cincinnati, Robert Gould Research Foundation, Inc., 1947.
8. Moore, Carl V.: Idiopathic hypochromic anemia—an obituary, Blood, Journal of Hematology 5:9 (Sept.) 1950.
9. Marlow, Arthur, and Taylor, F. L. H.: Constancy of iron in the blood plasma and urine in health and in anemia, Archives of Internal Medicine 53:71 (April) 1934.
10. Moore, Carl V.: Iron metabolism and hypochromic anemia, Symposia on Nutrition; Vol. I, Nutritional Anemia, Cincinnati, Robert Gould Research Foundation, Inc., 1947.
11. Darby, William J.: Iron and copper, J.A.M.A. 142:16 (April 22) 1950.
12. Grancik, S.: Iron metabolism and hemachromatosis, Bulletin New York Academy of Medicine (July) 1949.
13. Moore, C. V.; Arrowsmith, Wm. R.; Welch, J. O., and Minnich, Virginia: IV: Observations on the absorption of iron from the gastrointestinal tract, Studies in Iron Transportation and Metabolism, Journal of Clinical Investigation 18:5 (Sept.) 1939.
14. Dubach, Reubenia; Moore, C. V., and Minnich, Virginia: V: Utilization of intravenously injected radioactive iron for hemoglobin synthesis, and an evaluation of the radioactive iron method for studying iron absorption, Studies in Iron Transportation and Metabolism, Journal of Laboratory and Clinical Medicine 31:10 (Oct.) 1946.
15. Heath, Clark W.; Strauss, Maurice B., and Castle, William B.: Quantitative aspects of iron deficiency in hypochromic anemia, Journal of Clinical Investigation 9:11 (Nov.) 1932.
16. Hayden, Russel L.: The mechanism of anemia, Journal of Laboratory and Clinical Medicine 22:5 (Feb.) 1937.
17. Lund, Curtis J.: Studies of the iron deficiency anemia of pregnancy, American Journal of Obstetrics and Gynecology 62:5 (Nov.) 1951.
18. Wilson, Sloan J., and Boyle, Phyllis: Erroneous anemia and polycythemia, a comparative study of peripheral blood and blood volumes, A.M.A. Archives of Internal Medicine 90:5 (Nov.) 1952.
19. Thomson, M. L.: Hypochromic anemia in adolescent males, British Medical Journal (Oct. 9) 1943.
20. Moore, C. V.; Minnich, V.; Vilter, R. W., and Spies, T. D.: Hypochromic anemia in patients with deficiency of the vitamin B complex, J.A.M.A. 121:4 (Jan. 23) 1943.
21. Holly, Roy G.: Intravenous iron: evaluation of saccharated iron oxide in iron deficiency states in obstetrics and gynecology, Blood, Journal of Hematology 6:11 (Nov.) 1951.
22. Brown, E. B.; Moore, C. V.; Reynafarge, C., and Smith, D. E.: Intravenously administered saccharated iron oxide in the treatment of hypochromic anemia, J.A.M.A. 144:13.

ANNOUNCEMENTS

A regional meeting of the Central Region of the National Gastroenterological Association will be held in Milwaukee on March 28. Scientific sessions will be held at the Hotel Schroeder following the semi-annual meeting of the organization's council. Programs may be obtained from Dr. Joseph Shaiken, 536 West Wisconsin Avenue, Milwaukee 3, Wisconsin.

The Third Annual Seminar and Conference on Cancer Cytology will be held by the Cancer Institute at Miami, Florida, April 21-24. Information may be obtained from Cancer Research and Cytology Center, 1155 N.W. 14th Street, Miami, Florida.

The Fifth Annual Dr. F. G. Thompson, Sr., Lectureship will be presented at 902 Edmond Street, St. Joseph, Missouri, on Thursday, May 20, by Dr. Robert M. Moore, professor of surgery, University of Texas. Information may be secured from Thompson, Brumm and Knepper Clinic, St. Joseph, Missouri.

A meeting of the American Geriatrics Society will be held at the Fairmont Hotel, San Francisco, just preceding the meeting of the A.M.A. Scientific ses-



"...when the patient is in acute distress from waterlogging..."

"Meralluride sodium solution (MERCUHYDRIN) in 1 to 2 cc. doses intramuscularly has been very effective and is not painful."* In acute congestive failure, MERCUHYDRIN characteristically curbs tissue inundation and relieves dyspnea, orthopnea and cardiac asthma.

Ampuls of 1 cc., 2 cc., and 10 cc. vials.

*Stead, E. A., Jr., in Cecil, R. L., and Loeb, R. F.: Textbook of Medicine, ed. 8, Philadelphia, W. B. Saunders Co., 1951, p. 1065.

MERCUHYDRIN®
(meralluride injection U.S.P.)

Leadership in diuretic research
Lakeside LABORATORIES, INC., MILWAUKEE 1, WISCONSIN

sions will begin on June 17 and continue through June 19. The meeting will be open to all physicians interested in the field of geriatrics. Details may be secured from W. O. Thompson, M.D., 700 North Michigan Avenue, Chicago 11, Illinois.

The Third Annual Symposium on Tuberculosis and Other Chronic Pulmonary Diseases for General Practitioners will be held in Saranac Lake, New York, July 12-16. The course is approved by the American Academy of General Practice for 26 hours of credit. Programs and complete information may be secured from Chest Disease Symposium, P. O. Box 2, Saranac Lake, New York.

A select list of medical films available only to professional audiences may now be obtained free of charge upon request to the Princeton Film Center, Inc., Princeton, New Jersey. All are 16 mm. films, some silent, some with sound, many in color.

THE KANSAS PRESS LOOKS AT MEDICINE

Editor's Note. In this section the JOURNAL *reproduces editorials relating to medicine which have appeared in the lay press. An effort is made to include both favorable and unfavorable comments, and the Editorial Board in no instance assumes responsibility for the opinions expressed.*

On the Spot

Congressmen seem to be worried for fear President Eisenhower will in some way jeopardize their chances in the coming off-year election. And I think he may do just that, but not in the way they mean.

They are especially worried about his farm program and his health program. They are worried because he does not see fit to woo the pressure groups. And well they may be. But not because the pressure groups are so frightfully important. The danger to Congressmen who are up for election will come from the fact that Eisenhower's middle-of-the-road stand may put them on the spot and show their stripes to the voters.

For instance, Eisenhower's health program, like his farm program, was not cut to the measure of any pressure group. It was not designed to please the American Medical association nor yet the rafts of chronic dependents who bleed the country white in their demands for constant medical attention, some of it necessary and some of it not.

The President's proposal to underwrite voluntary health insurance groups was cut to the measure of the millions of small taxpayers who have contributed more than their share toward the nation's gigantic health relief bill and are still not wealthy enough to meet their own medical expenses without cruel hardship and sometimes bankruptcy. Already the American Medical association has expressed its displeasure over Eisenhower's proposal. But it has offered no substitute to that or to the bromide that the rich can pay their own bills and the poor can be taken care of.

Very soon our Congressmen will have an opportunity to vote for bills growing out of the chief executive's recommendation. If they vote against it they will please both the Medical Association and the organized reliefers. But they will infuriate hundreds of thousands of middle-class voters. And it was these voters, independents for the most part, who elected Eisenhower and who got many Congressmen into office on his shirt tails.

There are growing indications that Eisenhower proposes to fulfill his promise to serve as President of all Americans. Now the rank-and-file Americans have needed a President for a long time. If they see one in Eisenhower they will hang on to him. There is a possibility that Congressmen who thwart Eisenhower's efforts to serve the middle-class may get themselves licked to a finish without for a moment diminishing his popularity.—*Marion Ellet in Concordia Blade-Empire, January 23, 1954.*

Program of Kansas Council of Women

The Kansas Council of Women, in its proposed program of work for 1954, lists 18 objectives, three of which pertain to subjects of interest to the medical profession.

The first of the three is to "Continue participation in and support of statewide programs that encourage and strengthen protective health measures, safeguarding at all times state and local control of the programs."

Next is listed the support of legislation that will "(a) Continue to provide sufficient funds for adequate care and treatment of the mentally ill, and for training personnel; (b) improve commitment laws."

The third project pertains to work being carried on for psychotic, exceptional, and handicapped children, in addition to those living in Kansas institutions, and directs that the Kansas Council of Women co-operate with other recognized organizations to improve all forms of therapy and assistance.

Patronize JOURNAL advertisers.

innamon-flavored,

ready-mixed form of the new antibiotic

. . stable 18 months . . . administer any time



It's tasty. It's stable. It's *Pediatric* ERYTHROCIN Suspension—made especially for little patients. Rich in cinnamon flavor, *Pediatric* ERYTHROCIN has a sweet candy-like taste that children really like.



And it works. Against common winter coccal infections. Against pyoderma, erysipelas, and other infectious conditions. Especially advantageous against staphylococci—because of the high incidence of staphylococcal resistance to many other antibiotics and when the patient is allergically sensitive to other antibiotics.



Gastrointestinal disturbances rare. *Pediatric* ERYTHROCIN is specific in action—*less likely to alter normal intestinal flora than most other antibiotics.* No serious side effects reported.

Pediatric ERYTHROCIN comes in 2-fluidounce, pour-lip bottles. *No mixing required.* Can be administered before, after or with meals. Prescribe *Pediatric* ERYTHROCIN. Abbott

pediatric

Erythrocin

TRADE MARK

stearate

(Erythromycin Stearate, Abbott)

Oral Suspension

DOSAGE

One 5-cc. teaspoonful represents
100 mg. of ERYTHROCIN
25-lb. child—½ teaspoonful
50-lb. child—1 teaspoonful
100-lb. child—2 teaspoonfuls
Every 4 to 6 hours

1-95-54

BOOK REVIEWS

The Psychiatrist, His Training and Development, Report of the 1952 Conference on Psychiatric Education of the American Psychiatric Association and the Association of American Medical Colleges, June 19-25, 1952. Published by American Psychiatric Association, Washington 1, D. C., 1953. Pp. 214.

The focus of the study was primarily on the training of career psychiatrists, complementing the previous year's conference on psychiatry in undergraduate medical education, both of which were financed by the United States Public Health Institute. The purpose was the teaching of basic and clinical psychiatry and the coordinating of groups concerned with the problems of the use of total resources for the promotion and preservation of the emotional health of the community.

This book is a valuable historical document of current practices in residency training with well organized expression of opinions on their value and the reasons therefor. The transition from 1951 is logical and suggestive of the steps to come in the next edition. The scope of psychiatry is broadly considered both clinically and communally.

There is clear-cut recognition that the older eclectic methods of teaching are fast waning. In fact, the lecture method has given way to case demonstrations, group discussions, and to the almost universal method of personal supervision and individual teaching of each resident—the present method of choice.

The central theme of the informal discussion rested upon concern with the psychodynamic aspects of psychiatry. The development of a psychiatrist requires an understanding of personality in terms of psychodynamic concepts, requiring considerable time and effort in prolonged work with individual patients, far more than to gain adequate proficiency in many other aspects. What is taught as psychodynamics varies greatly in various centers. Hence, this conference attempted to lay dawn basic principles which were commonly believed to come in this category. (See Chapter 2.) In some centers it is merely neurology which is taught. In others, it is psychoanalytically oriented from the first day of residency.

The editors quote from Maxwell Gitelson, who wrote, "The chief contribution of psychoanalysis to American psychiatry has been to complete the structure which was originally given it by psychobiology. Without the concept of the unconscious and the things which follow from that concept, the 'sense and meaning' which Adolph Meyer observed in mental illness remained without system and unity. With this concept, psychiatry really has become holistic."

Between these points of view of teaching, Thomas A. C. Rennie says in his section on the teaching of dynamic psychiatry, "Many residents are too prone to consider that psychodynamics constitute all of psychiatry and to pursue their training largely with the aim of that practice in mind, to the exclusion of real concern with broad general competence and dynamic understanding of the wide range of psychiatric disorders. The fundamental aim of the residency program should be the attainment of such competence and understanding; within this framework, psychodynamics and psychotherapy are the major tools."

Much of this book is devoted to how this last may be accomplished and what it is all about. As one reads, one senses the conflicts which seem to be the central theme of this present generation in civilization. While it is not so written in this book, it is obvious that the discussors are well aware of the importance of the emotional maturation of the student

CLASSIFIED ADVERTISEMENTS

FOR SALE OR RENT—Fully equipped 3-room office, ground floor, instruments and medicines. Town of 800. Nearest competition 20 and 35 miles. Write the Journal 4-54.

FOR SALE—Office equipment, Picker x-ray, steel examining room set, examination chair, cabinets, lamps, fans, Alpine lamp, heat lamp, diathermy, maple reception furniture, oak office furniture, safe, instruments, microscope, complete well equipped office. Reasonable price. Optional—will rent eight-room brick office or sell on reasonable terms. Good location, county seat, large territory, first class city-owned hospital, surgeon needed, good will free. Retiring. Write the Journal 5-54.

WANTED—Registered medical technician, male or female, to work in a clinic of 12 doctors and in a midwestern city of 100,000 population. Excellent working conditions, no night calls. Write the Journal 2-54.

FOR SALE—Spencer monocular microscope, like new. Two eyepieces 10x and 6x; three objective lenses 10x, 44x and 95x oil immersion; mechanical stage: substage light condenser and individual lamp; carrying case. Excellent for medical student or physician's laboratory. Price $225. Write the Journal 3-54.

LOCATION WANTED. Will be released from service April 15, will begin residency October 1. Will assist or relieve physician during May, June, July, August. Formerly in general practice. K.U. graduate, member of Kansas Medical Society. Write the Journal 6-54.

FOR SALE—Portable x-ray (Profex), also permanent standard. Other equipment, all in good condition. Write the Journal 7-54.

Everything for the Laboratory

SOUTHWEST SCIENTIFIC CORPORATION

LABORATORY SUPPLIES AND EQUIPMENT

122 South St. Francis Street

Phone 2-0582 Wichita, Kansas

The Neurological Hospital

2625 West Paseo,

KANSAS CITY, MISSOURI

★ ★ ★

A voluntary hospital providing the care and treatment of nervous and mental patients and associate conditions.

SECLUSION MATERNITY



Write for information

MRS. EVA THOMSON
4911 East 27th St.
Kansas City, Mo.

FAIRMOUNT HOSPITAL

For Unmarried Girls
Est. 1909

Private sanitarium with certified obstetrician in charge. All adoptions arranged through juvenile court. Early entrance advised.

Rates reasonable. In certain cases work given to reduce expenses.

Nationally advertised Surgical Supplies and Equipment have been placed at Topeka, Joplin, Kansas City and St. Joseph for your convenience by—

GOETZE NIEMER COMPANY

Management by Dr. W. F. Goetze, member of the American Medical Ass'n, assures intelligent servicing of your orders.

UNIVERSITY OF KANSAS SCHOOL OF MEDICINE

Postgraduate Medical Courses

OPHTHALMOLOGY and OTOLARYNGOLOGY
April 5, 6, 7, 8 & 9

Guest Instructors:

FRANCIS HEED ADLER, M.D., Professor of Ophthalmology, University of Pennsylvania School of Medicine, Philadelphia.
FRANZ ALTMANN, M.D., Assistant Clinical Professor of Otolaryngology, Columbia University College of Physicians & Surgeons, New York City.
LEONARD CHRISTENSEN, M.D., Associate Professor of Ophthalmology, University of Oregon Medical School, Portland.
JEROME A. HILGER, M.D., Clinical Professor of Otolaryngology, University of Minnesota School of Medicine, Minneapolis.
WILLIAM F. HUGHES, JR., M.D., Professor of Ophthalmology, University of Illinois College of Medicine, Chicago.
JOSEPH H. OGURA, M.D., Associate Professor of Otolaryngology, Washington University School of Medicine, St. Louis, Mo.

Subjects to Be Discussed:

Differential Diagnosis in Iritis and Uveitis.
Optic Nerve Pathology (Fundus Photographs).
Diagnostic Examination in Strabismus.
Diagnosis and Management of Ptosis.
Gonioscopy.
Treatment of Iritis.
New Drugs in the Treatment of Glaucoma.
Neurological Fields.
Diabetic Changes in the Eye.
Pathological Studies of Ocular Surgery Complications.
Endolymphatic Hypertension and Its Treatment.
Facial Injury.
Physiology of the Labyrinthine Fluids.
Diseases of the Parotid Gland.
Reconstruction of the Nasal Pyramid—Part III.
Technique of Lateral Pharyngotomy.
Benign Tumors of the Larynx.
Pathogenesis of Perceptive Deafness.
Audiologic Techniques.
Etiology and Morbid Anatomy of Otosclerosis.

ANESTHESIOLOGY
April 12, 13 & 14

Guest Instructors:

VINCENT J. COLLINS, M.D., Director, Department of Anesthesiology, St. Vincent's Hospital, New York City.
STUART C. CULLEN, M.D., Professor of Surgery (Anesthesiology), State University of Iowa College of Medicine, Iowa City.
EDWIN J. DE BEER, Ph.D., Acting Director, Wellcome Research Laboratories, Tuckahoe, N. Y.
M. DIGBY LEIGH, M.D., Director, Department of Anesthesiology, Vancouver General Hospital, Vancouver, B. C.
JOHN S. LUNDY, M.D., Chairman, Section of Anesthesiology, The Mayo Clinic; Professor of Anesthesiology, University of Minnesota Graduate School of Medicine, Rochester.
O. SIDNEY ORTH, M.D., Professor of Pharmacology and Anesthesiology, and Chairman, Department of Anesthesiology, University of Wisconsin Medical School, Madison.
MAX S. SADOVE, M.D., Professor of Surgery and Head, Division of Anesthesiology, University of Illinois College of Medicine, Chicago.
SCOTT M. SMITH, M.D., Professor of Anesthesiology and Head of Department, University of Utah School of Medicine, Salt Lake City.
ELWYN L. CADY, JR., LL.B., Medicolegal Consultant; Director, Law-Medicine Program, University of Kansas City School of Law.

The course covers a wide variety of subjects—presented by didactic lectures, panel discussions and televised demonstrations—but emphasizes especially the problems of Anesthesia in Children, in the Cardiac Patient and in Traumatic Surgery; the Autonomic Nervous System and Endotracheal Anesthesia.

which must be enhanced if he is to achieve a reliable understanding of motives, the central purpose in psychodynamics. This is best achieved through the teacher-student relationship primarily.

Naturally, there was quite a discussion of the spontaneity of voluntary action versus determinism by environmental conditioning. The agreement was upon a considerable range of practical uncertainty in the prediction of behavior but general agreement as to the importance of events of childhood and social experience in shaping the general course of personality development and in the formation of value judgments.

One valuable field of discussion was the relation of psychiatrists to general staff members of the teaching centers. This emphasizes the great importance of finding a place for psychiatry in the general medical attitude. Those trained in the basic sciences walk easily from department to department without any sense of being out of their field as in pathology, medicine, surgery, pediatrics, etc. As yet, basic science is just now including the science of human behavior and ultimately this estrangement, between psychiatrist and the general staff, one from the other, should become gradually lessened.

The book is a thoughtful one with a good collection of factual data as guides for decision in the resident program.—*S. A.*

Cure Your Nerves Yourself. By Louis E. Bisch. Published by Wilfred Funk, Inc., New York, New York. 247 pages. Price $3.50.

Some retail merchants attribute their success to the fact that they concentrate on selling articles that people really need or want. At the same time, they attempt to dissuade customers from buying useless or inappropriate goods. That principle, applied to psychiatry, is outlined in this book.

The author, who has devoted himself to psychiatry and neurology for 42 years, succeeds admirably in accomplishing the three objectives he lists for his book: (1) To prove to you that most cases of nerves do not require professional psychiatric treatment; (2) To help you discover for yourself if yours is such a case; (3) To show you how you can cure yourself.

The style is easy and informal, and nomenclature that would be confusing to a layman is omitted. The book can be recommended to any patient who would benefit from a quiet self-appraisal. Although it is designed to help those who feel that they may need psychiatric care, it would be beneficial to all who find themselves blaming "nerves" for the troubles that beset them.

Patronize JOURNAL advertisers.

It's Convenient to Buy Kanox

THERAPEUTIC OXYGEN AND ANESTHETIC GASES

Oxygen Therapy

Regulators and Equipment

Repair Service for

Regulators, Flowmeters

and Humidifiers



Nitrous Oxide

Ethylene

Carbon Dioxide

Helium

Oxygen

Cyclopropane

KANSAS OXYGEN, Inc.

Phone 5-5551 HUTCHINSON, KANSAS Box 551

Deformity Appliances of Quality

Orthopedic and Surgical Appliances

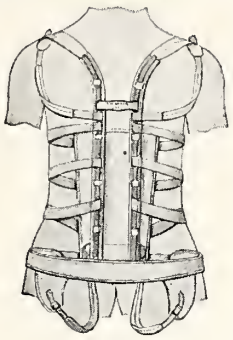
Artificial Limbs
Trusses

Abdominal Supports

Elastic Hosiery

Foot Supports

Surgical Corsets



Taylor Back Brace
Made to Order in
Our Own Factory

P. W. HANICKE MFG. CO.
1009 McGee St. Victor 4750
KANSAS CITY, MO.

Medical and Surgical Supplies for Doctors of Medicine and Hospitals

Munns Medical Supply Co.
512 Kansas Avenue
Topeka, Kansas

ACCIDENT, HOSPITAL AND SICKNESS INSURANCE

For Physicians, Surgeons and Dentists Exclusively

ALL PREMIUMS COME FROM — PHYSICIANS SURGEONS DENTISTS — ALL CLAIMS GO TO

$5,000 accidental death Quarterly $8.00 **$25 weekly indemnity, accident and sickness**	**$15,000 accidental death Quarterly $24.00** **$75 weekly indemnity, accident and sickness**
$10,000 accidental death Quarterly $16.00 **$50 weekly indemnity, accident and sickness**	**$20,000 accidental death Quarterly $32.00** **$100 weekly indemnity, accident and sickness**

Cost Has Never Exceeded Amounts Shown

ALSO HOSPITAL INSURANCE

	Single	*Double*	*Triple*	*Quadruple*
60 days in Hospital	5.00 per day	10.00 per day	15.00 per day	20.00 per day
30 days of Nurse at Home	5.00 per day	10.00 per day	15.00 per day	20.00 per day
Laboratory Fees in Hospital	5.00	10.00	15.00	20.00
Operating Room in Hospital	10.00	20.00	30.00	40.00
Anesthetic in Hospital	10.00	20.00	30.00	40.00
X-Ray in Hospital	10.00	20.00	30.00	40.00
Medicines in Hospital	10.00	20.00	30.00	40.00
Ambulance to or from Hospital	10.00	20.00	30.00	40.00
COSTS (Quarterly)				
Adult	2.50	5.00	7.50	10.00
Child to age 19	1.50	3.00	4.50	6.00
Child over age 19	2.50	5.00	7.50	10.00

$4,000,000.00 INVESTED ASSETS

PHYSICIANS CASUALTY ASSOCIATION
PHYSICIANS HEALTH ASSOCIATION

$19,500,000.00 PAID FOR CLAIMS

51 years under the same management

400 First National Bank Building Omaha 2, Nebraska

$200,000.00 deposited with State of Nebraska for protection of our members

TV Guide Takes Medical Viewpoint

A recent *TV Guide* magazine editorial upheld a viewpoint long held by the medical profession with regard to the CBS-TV Strike It Rich program. It started by saying:

"It must be a cinch to write for the Daily Worker, Pravda and other Communist sheets as long as TV press agents dish out publicity releases like this one:

" 'Even though she failed to win the top cash prize on the CBS-TV Strike It Rich program, Mrs. ———, of Philadelphia, "struck it rich" . . . when she appeared as a recent contestant. In desperate need of medical aid, Mrs. ——— won $115 toward it, but through the show's heart line telephone, she received an additional $100, plus free medical treatment for as long as she may need it.' "

Then the magazine's editorial said:

"Can't you just hear the Red propagandists citing that as proof that the poor in America must bare their misery on television shows in the hope of winning medical treatment?

"The fact is that the poor in America receive medical care as good as, if not better than, anyone else. Leading doctors give unstintingly of their time, and hospitals and clinics of their facilities, so that those who require treatment and can't afford it can be helped—without charge. No one, especially in a great medical center such as Philadelphia, has to be 'in desperate need of medical aid.' No one has to appear on television to ask for such aid.

"In all fairness to Strike It Rich, the program did not originate the idea of using human misery as a form of entertainment. Nero, for one, had a great time with it. If, today, a television show can thrive on misery, and if that's what viewers want to see, the sponsors of the show have good reason to keep it on the air.

"But we can, and do, object strenuously to stupid publicity material that presents a false picture of our way of life in order to grab off a few lines of newspaper or magazine space."

The incidence of tuberculosis is much higher among medical students than among similar groups of young adults in the general population. The primary complex is not necessarily benign. There is much evidence that it may directly precede progressive pulmonary tuberculosis or any complication, and therefore a re-evaluation of so-called minimal or benign disease seems necessary.—*William A. Abruzzi, Jr., M.D., and Rufus J. Hummel, M.D., The New England Journal of Medicine, April 23, 1953.*



Woodcroft Hospital--Pueblo, Colorado

A private hospital for the scientific treatment of neuro-psychiatric disorders, including alcoholism and drug addiction. Beautiful landscaping and home-like surroundings afford a restful atmosphere. Accommodations vary from single rooms with or without bath to rooms en suite, allowing for segregation of guests.

Detailed information furnished on request.

KARL J. WAGGENER, M.D.

ıtstanding therapeutic agents

Chloromycetin®

(Chloramphenicol, Parke-Davis)

ground which singles out and gives recognition to that product's place in the practice of medicine.

More than 11,000,000 patients have been treated with CHLOROMYCETIN. Today its vast "proving ground" reaches out and extends into practically every country of the civilized world.



Parke, Davis & Company

DETROIT 32, MICHIGAN

TABLE OF CONTENTS

APRIL, 1954

OFFICIAL PROGRAM, 95TH ANNUAL SESSION

Guest Speakers 188
Schedule of Events 194
Eye, Ear, Nose, and Throat Section . . . 198
Special Events 199
President's Page 206
Councilor Reports 207
Kansas Blue Shield 209
Kansas State Advisory Committee 210
Committee Reports 211

Entered as second-class matter at the post office at Fulton, Missouri, June 19, 1953, under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of October 3, 1917, authorized on July 2, 1918. Office of Publication: 1201-05 Bluff Street, Fulton, Missouri.

THE JOURNAL OF THE KANSAS MEDICAL SOCIETY

Editorial Board

ORVILLE R. CLARK, M.D., *Editor,* Topeka
JOHN W. CAVANAUGH, M.D., Topeka
DAVID E. GRAY, M.D., Topeka
RICHARD GREER, M.D., Topeka
DWIGHT LAWSON, M.D., Topeka

Associate Editors

DONALD P. TREES, M.D., Wichita
GLEN R. SHEPHERD, M.D., Kansas City

Information

Ownership: The Journal is a non-profit publication owned and published monthly by the Kansas Medical Society.

Subscription: A year's subscription to the Journal is included in membership in the Kansas Medical Society, with $2.00 of each member's dues apportioned to the Journal. Rates to others, except in foreign countries, $4.00 per year or 60c per copy.

Material: Scientific articles, editorials, and data of general interest are invited from all members. Articles are to be submitted on condition that they are contributed solely to this publication. A right is reserved to reject any material deemed unsatisfactory.

Manuscripts: Only manuscripts that are typewritten on one side, double spaced, and original copies can be accepted. Manuscripts will be returned upon request.

Advertising: All advertising contracts, and all copy from advertisers under contract are subject to approval of the editorial board. Copy should be received by the 20th of the month immediately preceding the month of publication.

Non-Responsibility: Although an effort is made to publish only accurate articles and legitimate advertisements, the Journal denies legal responsibility for any statements, opinions, or advertisements appearing under the names of contributors or concerns.

PAULINE FARRELL, Managing Editor and Advertising Manager

OLIVER E. EBEL, Business Manager

THE KANSAS MEDICAL SOCIETY

President.....LUCIEN R. PYLE, M.D., Topeka
Past President....WARREN F. BERNSTORF, M.D., Winfield
President-Elect.....MURRAY C. EDDY, M.D., Hays
First Vice-President..JOHN M. PORTER, M.D., Concordia
Second Vice-President..CLYDE W. MILLER, M.D., Wichita
Constitutional Secretary..DALE D. VERMILLION, M.D., Goodland
Treasurer.....JOHN L. LATTIMORE, M.D., Topeka
AMA Delegate 1953-1954, LAURENCE S. NELSON, M.D., Salina
AMA Delegate 1954-1955, GEORGE F. GSELL, M.D., Wichita

Councilors and Councilor Districts

First District.....W. L. ANDERSON, M.D., Atchison
Second District .....H. P. JONES, M.D., Lawrence
Third District....CHARLES E. VESTLE, M.D., Humboldt
Fourth District.....F. C. TAGGART, M.D., Topeka
Fifth District.....HAROLD M. GLOVER, M.D., Newton
Sixth District.....NORTON L. FRANCIS, M.D., Wichita
Seventh District..SEVERT A. ANDERSON, M.D., Clay Center
Eighth District.....A. E. RUEB, M.D., Salina
Ninth District.....JAMES L. JENSON, M.D., Colby
Tenth District.....L. W. REYNOLDS, M.D., Hays
Eleventh District.....C. V. BLACK, M.D., Pratt
Twelfth District.....R. G. KLEIN, M.D., Dodge City

OLIVER E. EBEL, Executive Secretary

RUEBEN M. DALBEC, Executive Assistant

Executive Office: 315 West Fourth Street, Topeka

THE JOURNAL
of the
KANSAS MEDICAL SOCIETY

Copyright, 1954, by The Kansas Medical Society

Volume LV APRIL, 1954 No. 4

Greetings!

We of the Shawnee County Medical Society consider it a privilege and an honor to be hosts to the Kansas Medical Society for the annual meeting, May 2-6, 1954.

Our committees, under the able supervision of Dr. Charles S. Joss as general chairman, have worked hard to prepare a program which, though differing somewhat from those in the past, we feel will be of value and interest to all members of the Society.

The Woman's Auxiliary to the Shawnee County Medical Society has also formed an interesting and entertaining program for the ladies, so there will be activities for all.

The annual banquet, which will be held this year at the new Topeka Country Club, will be a must for excellent food and entertainment.

It would be wise to contact the hotel of your choice as early as possible for your reservations so as to attend and enjoy all phases of the four days of activities planned.

Just a reminder too that our medical assistants have their annual meeting May 2 and 3. They are trying to do a big and very important job and need our support. Please encourage your assistant to attend the business and social activities which the Shawnee County chapter has planned for them.

The scientific sessions and exhibits will be in the Municipal Auditorium, and the noon luncheons will be alternated between the Hotels Jayhawk and Kansan.

Plan to come, to learn, to meet old and new friends, and to return to your respective communities refreshed after what we feel will be a stimulating program.

Fraternally yours,

FRANCIS T. COLLINS, M.D., *President*
Shawnee County Medical Society

Guest Speakers

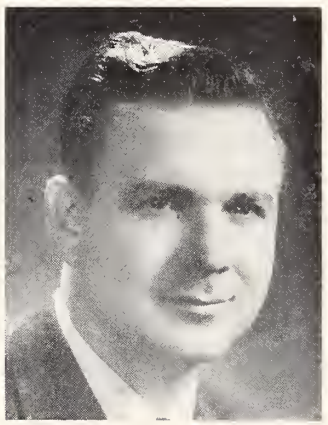


DAVID R. AKERS, M.D.

Denver, Colorado

Graduate, University of Colorado School of Medicine, 1936; Assistant Clinical Professor of Surgery, University of Colorado Medical Center; Assistant Chief of Surgical Service, Children's Hospital, Denver; Diplomate, American Board of Surgery; Fellow, American College of Surgeons; Member, Southwestern Surgical Congress and Denver Academy of Surgery.

Specialty: Surgery.



FREDERIC G. BURKE, M.D.

Washington, D. C.

Graduate, Georgetown University School of Medicine, 1942; Professor of Pediatrics and Director of Department of Pediatrics, Georgetown University Medical Center; Principal Investigator, Children's Hospital Research Foundation; Chief of Staff, Children's Convalescent Home, Washington; Member of Editorial Board, *Clinical Proceedings of Children's Hospital* and *Medical Annals of the District of Columbia;* Diplomate, American Board of Pediatrics; Fellow, American Academy of Pediatrics.

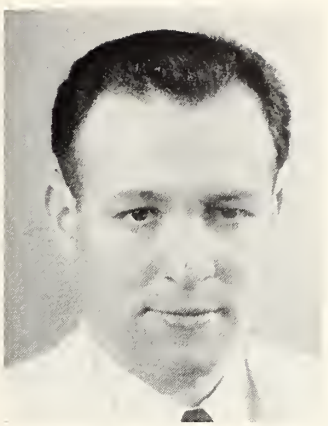
Specialty: Pediatrics.

JAMES R. COOK, M.D.

Cleveland, Ohio

Graduate, Indiana University School of Medicine, 1944; Consultant, Division of Medicine and Endocrinology, Cleveland Clinic; Instructor in Medicine, Bunts Institute; Diplomate, American Board of Internal Medicine; Member, American Diabetes Association and Central Society for Clinical Research.

Specialty: Internal Medicine.



OLIVER COPE, M.D.

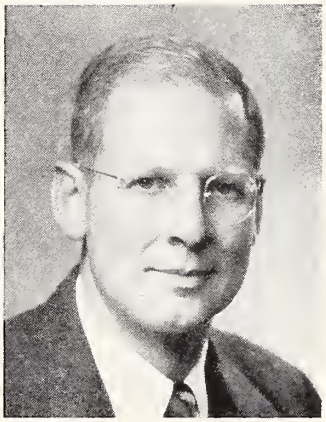
Boston, Massachusetts

Graduate, Harvard Medical School, 1928; Associate Professor of Surgery, Harvard Medical School; Visiting Surgeon, Massachusetts General Hospital; Doctor Honoris Causa, University of Toulouse, France, 1950; Diplomate, American Board of Surgery; Fellow, American College of Surgeons; Member, American Surgical Association, Society of Clinical Surgery, American Society for Clinical Investigation, and Society of University Surgeons.

Specialty: Surgery.



TELEPHONE NUMBER AT THE AUDITORIUM—9-9944



CHARLES E. ILIFF, M.D.

Baltimore, Maryland

Graduate, Johns Hopkins University School of Medicine, 1939; Assistant Professor of Ophthalmology, Johns Hopkins University; Ophthalmologist-in-Charge, Johns Hopkins Hospital; Consultant in Ophthalmology, Anne Arundel General Hospital; Diplomate, American Board of Ophthalmology; Fellow, American College of Surgeons and American Academy of Ophthalmology and Otolaryngology.

Specialty: Ophthalmology.



ORMAND C. JULIAN, M.D.

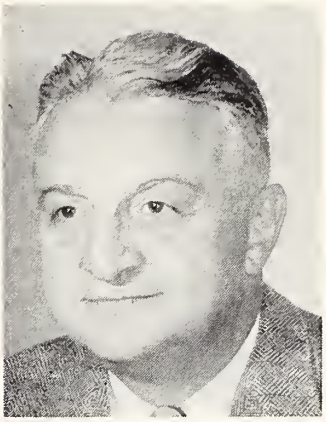
Chicago, Illinois

Graduate, University of Chicago, Division of Biological Sciences, 1937; Associate Professor of Surgery, University of Illinois College of Medicine; Associate Attending Surgeon, St. Luke's Hospital, Chicago; Diplomate, American Board of Surgery; Fellow, American College of Surgeons; Member, Central Surgical Association, Western Surgical Association, Society for Vascular Surgeons, and International Society of Angiology.

Specialty: Cardiovascular Surgery.

ANDREW A. MARCHETTI, M.D.

Washington, D. C.



Graduate, Johns Hopkins University School of Medicine, 1928; Professor of Obstetrics and Gynecology, Georgetown University School of Medicine; Consultant, Walter Reed Army Medical Center; Diplomate, American Board of Obstetrics and Gynecology; Fellow, American Academy of Obstetrics and Gynecology, American Association of Obstetricians, Gynecologists and Abdominal Surgeons, and American College of Surgeons; Member, American Gynecological Society.

Specialty: Obstetrics and Gynecology.

PAUL M. MOORE, M.D.

Cleveland, Ohio



Graduate, University of Michigan Medical School, 1922; Associate Surgeon, Department of Otolaryngology, Huron Road Hospital; Assistant Surgeon, Department of Otolaryngology, St. Luke's Hospital; Diplomate, American Board of Otolaryngology; Member, American Academy of Ophthalmology and Otolaryngology, American Laryngological, Rhinological and Otological Society, American Bronchoesophagological Association, and Pan American Association of Otorhinolaryngology and Bronchoesophagology.

Specialty: Otolaryngology.

TELEPHONE NUMBER AT THE AUDITORIUM—9-9944

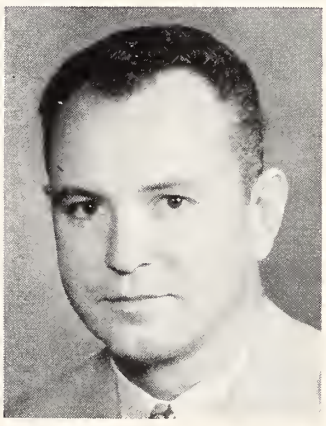


SHEPARD SHAPIRO, M.D.

New York, New York

Graduate, New York University College of Medicine, 1919; Assistant Professor of Clinical Medicine, New York University College of Medicine; Visiting Physician, Coldwater Memorial Hospital and Lincoln Hospital; Associate Visiting Physician, University Hospital; Conducted Early Investigation of Dicumarol in Man; Introduced Warfarin and Warfarin Sodium to Profession in 1953, after Research Work with Discoverer, Professor K. P. Link.

Specialty: Internal Medicine.



MAURICE H. STAUFFER, M.D.

Rochester, Minnesota

Graduate, University of Kansas School of Medicine, 1941; Consultant in Medicine, Mayo Clinic; Instructor in Medicine, Mayo Foundation, University of Minnesota; Diplomate, American Board of Internal Medicine; Member, American Association for the Study of Liver Diseases.

Specialty: Gastroenterology.

BERNARD A. WATSON, M.D.

Clifton Springs, New York

Graduate, University of Michigan Medical School, 1929; Director, Division of Medicine, Clifton Springs Sanitarium and Clinic; Instructor in Medicine, University of Rochester School of Medicine; Diplomate, American Board of Internal Medicine; Fellow, American College of Physicians; Member, American Diabetes Association, Association for the Study of Internal Secretions, and Society of Medical Consultants to the Armed Forces.

Specialty: Internal Medicine.



Annual Meeting Committees

General Chairman, Charles S. Joss, M.D.

AUXILIARY

Robert E. Pfuetze, M.D., Floyd C. Taggart, M.D.

BANQUET

Richard H. Greer, M.D., Howard U. Kennedy, M.D.

COMMERCIAL EXHIBITS

Floyd C. Beelman, M.D., Shirley E. Clark, M.D., Donald H. Macrae, M.D.

E.E.N.T. PROGRAM

Harold W. Powers, M.D.

GOLF

Byron J. Ashley, M.D.

LUNCHEON ARRANGEMENTS

Clarence K. Schaffer, M.D., Henry S. Blake, M.D.

MEDICAL ASSISTANTS

Harry J. Davis, M.D.

PUBLICITY

David E. Gray, M.D., William O. Martin, M.D.

SCIENTIFIC EXHIBITS

Robert R. Woods, M.D., Homer L. Hiebert, M.D., A. A. Fink, M.D.

SCIENTIFIC PROGRAM

Daniel L. Tappen, M.D., Orville R. Clark, M.D., Homer L. Hiebert, M.D., and Newman V. Treger, M.D., all of Topeka; Stanley L. VanderVelde, M.D., and Philip W. Morgan, M.D., both of Emporia.

SKEET SHOOT

R. Dale Dickson, M.D., Walter Mau, M.D.

TELEPHONE NUMBER AT THE AUDITORIUM—9-9944

Schedule of Events

95th Annual Session

Topeka, Kansas, May 2, 3, 4, 5, 6, 1954

Sunday, May 2

11:00 Annual Meeting, Board of Directors, Kansas Physicians' Service

Jayhawk Hotel, Rose Room

Monday Morning, May 3

9:30 Business Session, Kansas Chapter, American Academy of General Practice

See Page 200

10:00 Practice Rounds, Kansas Medical Golfing Association, Lunch Available

Topeka Country Club, 27th and Buchanan Streets

10:00 Practice Shooting, Kansas Medical Skeet and Trapshooting Association

Topeka Gun Club, Six Miles East of Topeka on Highway 40, North Side of Highway

Monday Afternoon, May 3

1:00 Competitive Golfing, Kansas Medical Golfing Association

Topeka Country Club, 27th and Buchanan Streets

1:00 Competitive Shooting, Kansas Medical Skeet and Trapshooting Association

Topeka Gun Club, Six Miles East of Topeka on Highway 40, North Side of Highway

1:30 Scientific Session, Kansas Chapter, American Academy of General Practice

See Page 200

3:00 Viewing of Exhibits at Muncipal Auditorium by Kansas Medical Assistants' Society

Monday Evening, May 3

6:00 Cocktails

Topeka Country Club, 27th and Buchanan Streets

6:30 Symposium and Banquet, Kansas Chapter, American Academy of General Practice

See Page 200

7:30 Tournament Banquet, Entertainment, Awarding of Prizes

Topeka Country Club, 27th and Buchanan Streets

Guest Speakers (showing films) from the University of Kansas—Phog Allen, Basketball Coach, and Chuck Mather, Football Coach

TELEPHONE NUMBER AT THE AUDITORIUM—9-9944

Tuesday Morning, May 4

8:00 Breakfast, Blue Shield Physician Relations Committee
Jayhawk Hotel, Green Room A

8:00 Registration
Municipal Auditorium, South Entrance
Open 8:00 a.m. to 5:00 p.m.
Opening of Scientific and Technical Exhibits

9:00 Business Session and Program, Kansas Association of Clinic Managers
See Page 202

FIRST GENERAL SESSION

Francis T. Collins, M.D., Topeka, *Presiding*

9:15 Francis T. Collins, M.D., Topeka, President, Shawnee County Medieal Society

Address of Welcome

9:30 Bernard A. Watson, M.D., Clifton Springs

The Offiee Management of Diabetes

10:05 Oliver Cope, M.D., Boston

Hyperparathyroidism: Experience with 152 Cases in 20 Years

10:35 Intermission to Visit Exhibits

SECOND GENERAL SESSION

Charles S. Joss, M.D., Topeka, *Presiding*

10:50 David R. Akers, M.D., Denver

Intussusception

11:25 Ormand C. Julian, M.D., Chieago

The Surgieal Treatment of Major Disease of the Abdominal Aorta

LUNCHEON SESSION

12:15 Jayhawk Hotel, Roof

Orville R. Clark, M.D., Topeka, *Presiding*

Tuesday Afternoon, May 4

THIRD GENERAL SESSION

2:00 Panel on Peripheral Vaseular Disease

Max S. Allen, M.D., Kansas City, *Moderator*

Ormand C. Julian, M.D., Chicago

Shepard Shapiro, M.D., New York

3:20 Intermission to Visit Exhibits

FOURTH GENERAL SESSION

3:40 Panel on Pre- and Postoperative Care of Infants and Children

Frank L. Menehan, M.D., Wichita, *Moderator*

David R. Akers, M.D., Denver

Frederic G. Burke, M.D., Washington

4:00 Meeting, Kansas Chapter, American College of Chest Physicians. See Page 202

4:00 Meeting, Kansas Society of Anesthesiologists
Place to be Announced

Tuesday Evening, May 4

5:00 Meeting, Cocktails, Dinner, Kansas State Pediatric Society
Topeka Country Club, 27th and Buchanan Streets

5:30 Meeting, Cocktails, Dinner, Kansas State Obstetrical Society
Kansan Hotel, Walnut and Indian Rooms

6:30 House of Delegates Dinner and Meeting
Jayhawk Hotel, Florentine Room

TELEPHONE NUMBER AT THE AUDITORIUM—9-9944

Wednesday Morning, May 5

7:30 Breakfast, Kansas Radiological Society
Jayhawk Hotel, Green Room C

7:30 Breakfast, Kansas Society of Pathologists
Jayhawk Hotel, Green Room A

8:30 Registration
Municipal Auditorium, South Entrance
Open 8:30 a.m. to 5:00 p.m.
Opening of Scientific and Technical Exhibits

FIFTH GENERAL SESSION

R. Dale Dickson, M.D., Topeka, *Presiding*

9:30 David R. Akers, M.D., Denver

Surgical Emergencies in the Newborn

10:05 Oliver Cope, M.D., Boston

An Appraisal of Radioactive Iodine in the Diagnosis and Treatment of Thyroid Disease

10:35 Intermission to Visit Exhibits

SIXTH GENERAL SESSION

Robert H. O'Neil, M.D., Topeka, *Presiding*

10:50 Frederic G. Burke, M.D., Washington

The Diagnosis and Treatment of Bacterial Meningitis in Infants and Children

11:25 Maurice H. Stauffer, M.D., Rochester

Jaundice: Clinical and Laboratory Clues in Diagnosis and Management

LUNCHEON SESSION

12:15 Kansan Hotel, Roof

Floyd C. Taggart, M.D., Topeka, *Presiding*

Wednesday Afternoon, May 5

SEVENTH GENERAL SESSION

2:00 Panel on Metabolic and Thyroid Disease

Edward H. Hashinger, M.D., Kansas City, *Moderator*

James R. Cook, M. D., Cleveland

Oliver Cope, M.D., Boston

Bernard A. Watson, M.D., Clifton Springs

3:20 Intermission to Visit Exhibits

EIGHTH GENERAL SESSION

3:40 Panel on Uterine Inertia and Prolonged Labor

Robert G. Heasty, M.D., Manhattan, *Moderator*

Hubert M. Floersch, M.D., Kansas City

Andrew A. Marchetti, M.D., Washington

Wednesday Evening, May 5

ANNUAL BANQUET

7:00 Topeka Country Club, 27th and Buchanan Streets

Lucien R. Pyle, M.D., Topeka, *Presiding*

Music by Pauline Deniston, Kansas City, and Vera McNary and her Strata Marimba, Kansas City

Dancing to the Music of Johnny Coon and His Orchestra

TELEPHONE NUMBER AT THE AUDITORIUM—9-9944

Thursday Morning, May 6

8:30 Registration
Municipal Auditorium, South Entrance
Open 8:30 a.m. to 12:00 Noon
Opening of Scientific and Technical Exhibits

NINTH GENERAL SESSION

NATHANIEL UHR, M.D., Topeka, *Presiding*

9:30 MAURICE H. STAUFFER, M.D., Rochester

Upper Abdominal Pain in Patients with Negative X-ray Studies

10:05 SHEPARD SHAPIRO, M.D., New York

Clinical Experiences with Cumidin Sodium (Warfarin Sodium), a New Hypoprothrombinemia-Inducing Agent

10:45 Intermission to Visit Exhibits

TENTH GENERAL SESSION

DANIEL L. TAPPEN, M.D., Topeka, *Presiding*

10:50 JAMES R. COOK, M.D., Cleveland

Diabetic Complications

11:25 ANDREW A. MARCHETTI, M.D., Washington

Use and Application of Forceps

LUNCHEON SESSION

12:30 House of Delegates Luncheon and Meeting

Jayhawk Hotel, Florentine Room

Technical Exhibits

1. W. B. Saunders Company
2. Goetze-Niemer Company
3. International Business Machines Corporation
4. Warner Chilcott Laboratories
5. Blue Cross-Blue Shield
6. General Electric Company, X-Ray Department
7. Winthrop-Stearns, Inc.
8, 9, 10. Munns Medical Supply Company
11. H. G. Fischer & Company
13. Physical Medicine Company
16. Hoffmann-La Roche, Inc.
17. Wm. P. Poythress & Company, Inc.
18. Washington National Insurance Company
19. S. E. Massengill Company
20. Eli Lilly and Company
21. Doho Chemical Corporation
22. Parke, Davis & Company
23. J. B. Lippincott Company
24. American Optical Company
25. Encyclopedia Americana
26 and 27. Mid-West Surgical Supply Company, Inc.
28. The Medical Protective Company
29. Bilhuber-Knoll Corporation
30. Coe Surgical Supply Company
31. Burroughs Wellcome & Co. (U.S.A.) Inc.
32. Greb X-ray Company
33. Abbott Laboratories
34. Carroll Dunham Smith Pharmacal Company
35. C. B. Fleet Company, Incorporated
36. Wyeth Laboratories
37. Ethicon Suture Laboratories, Incorporated
38. Ayerst, McKenna & Harrison, Ltd.
39. Pet Milk Company
40. Sharp & Dohme
41. Schering Corporation
42. Ciba Pharmaceutical Products, Inc.
43. Sandoz Chemical Works, Incorporated
44. Coufal-Kelcket X-Ray Company
46. Lederle Laboratories Division, American Cyanamid Company
47. A. S. Aloe Company
48. Commercial Insurance Company
49. Gerber Products Company
50. Mead Johnson & Company
51. M & R Laboratories
52. U. S. Vitamin Corporation
53. Quinton-Duffens Optical Company
54. The National Drug Company
55. Ortho Pharmaceutical Corporation
56. Filter Queen of Kansas
57. Pfizer Laboratories
58. United Medical Equipment Company
59. A. H. Robins Company, Inc.
60. The Baker Laboratories, Inc.
61. Thomas A. Edison, Incorporated
62. J. B. Roerig and Company
66. E. R. Squibb & Sons
67. The Wm. S. Merrell Company
68. G. D. Searle & Company
The Coca-Cola Company

Eye, Ear, Nose, and Throat Section

Tuesday Morning, May 4

8:00 Registration
Municipal Auditorium, South Entrance
Open 8:00 A.M. to 5:00 P.M.

FIRST SESSION

NORTON L. FRANCIS, M.D., Wichita, *Presiding*

9:30 PAUL M. MOORE, M.D., Cleveland

Eustachian Tube Problems

10:30 Intermission to Visit Exhibits

SECOND SESSION

KARL W. STOCK, M.D., Topeka, *Presiding*

11:00 CHARLES E. ILIFF, M.D., Baltimore

Surgery of the Orbit

12:15 Luncheon, Kansan Hotel, Roof Lounge
GEORGE F. GSELL, M.D., Wichita, *Presiding*

Tuesday Afternoon, May 4

THIRD SESSION

CHARLES R. KEMPTHORNE, M.D., Manhattan, *Presiding*

2:00 PAUL M. MOORE, M.D., Cleveland

Otitis Externa

3:00 Intermission to Visit Exhibits

FOURTH SESSION

ALBERT N. LEMOINE, JR., M.D., Kansas City, *Presiding*

3:30 CHARLES E. ILIFF, M.D., Baltimore

Dacryocystorhinostomy

4:30 Business Meeting

Wednesday Morning, May 5

8:30 Registration
Municipal Auditorium, South Entrance
Open 8:30 A.M. to 5:00 P.M.

FIFTH SESSION

G. O. PROUD, M.D., Kansas City, *Presiding*

9:30 PAUL M. MOORE, M.D., Cleveland

Rehabilitation of the Laryngectomized Patient

10:30 Intermission to Visit Exhibits

SIXTH SESSION

JAMES H. ENNS, M.D., Newton, *Presiding*

11:00 CHARLES E. ILIFF, M.D., Baltimore

Ptosis Surgery

12:15 Luncheon, Kansan Hotel, Roof Lounge
GEORGE F. GSELL, M.D., Wichita, *Presiding*

Wednesday Afternoon, May 5

SEVENTH SESSION

H. L. KIRKPATRICK, M.D., Topeka, *Presiding*

2:00 PAUL M. MOORE, M.D., Cleveland

Lesions of the Maxillary Sinus

3:00 Intermission to Visit Exhibits

EIGHTH SESSION

MAX S. LAKE, M.D., Salina, *Presiding*

3:15 CHARLES E. ILIFF, M.D., Baltimore

Cataract Surgery

TELEPHONE NUMBER AT THE AUDITORIUM—9-9944

Special Events

FOR ALL PHYSICIANS

Sportsmen's Events—Monday, May 3. Golf at 10:00 and 1:00, Topeka Country Club, 27th and Buchanan; Shooting at 10:00 and 1:00, Topeka Gun Club, Six Miles East of Topeka on Highway 40, North Side of Highway; Tournament Banquet at 7:30, Topeka Country Club, 27th and Buchanan

Round Table Luncheons—Tuesday, May 4, 12:15, Jayhawk Hotel, Roof, and Wednesday, May 5, 12:15, Kansan Hotel, Roof

Annual Banquet—Wednesday, May 5, 7:00, Topeka Country Club, 27th and Buchanan

FOR GROUPS

Anesthesiologists

Kansas Society of Anesthesiologists Meeting—Tuesday, May 4, 4:00, Place to be Announced

Blue Shield

Blue Shield Physician Relations Committee Breakfast and Meeting—Tuesday, May 4, 8:00, Jayhawk Hotel, Green Room A

Chest Physicians

Kansas Chapter, American College of Chest Physicians Meeting and Dinner—Tuesday, May 4, 4:00, Kansan Hotel, Walnut Room

Council

First Meeting of New Council—Thursday, May 6, following House of Delegates Meeting, Jayhawk Hotel, Florentine Room

E.E.N.T.

Kansas E.E.N.T. Society Business Meeting—Tuesday, May 4, 4:30, following E.E.N.T. Scientific Program; Luncheons—Tuesday, May 4, 12:15, and Wednesday, May 5, 12:15, Kansan Hotel, Roof Lounge

General Practitioners

Kansas Chapter, American Academy of General Practice—Monday, May 3, All through Day, Jayhawk Hotel

House of Delegates

Dinner Meeting—Tuesday, May 4, 6:30, Jayhawk Hotel, Florentine Room. Luncheon Meeting—Thursday, May 6, 12:30, Jayhawk Hotel, Florentine Room

Kansas Physicians' Service

Annual Meeting, Board of Directors—Sunday, May 2, 11:00, Jayhawk Hotel, Rose Room

Obstetricians

Kansas State Obstetrical Society Meeting, Cocktails, Dinner—Tuesday, May 4, 5:30, Kansan Hotel, Walnut and Indian Rooms

Pathologists

Kansas Society of Pathologists Breakfast and Meeting—Wednesday, May 5, 7:30, Jayhawk Hotel, Green Room A

Pediatricians

Kansas State Pediatric Society Meeting, Cocktails, Dinner—Tuesday, May 4, 5:00, Topeka Country Club, 27th and Buchanan

Radiologists

Kansas Radiological Society Breakfast and Meeting—Wednesday, May 5, 7:30, Jayhawk Hotel, Green Room C

Vocational Rehabilitation

Vocational Rehabilitation Service, Professional Advisory Committee Breakfast and Meeting, Tuesday, May 4, 7:45, Jayhawk Hotel, Rose Room

Kansas Chapter, American Academy of General Practice

ANNUAL ASSEMBLY

Jayhawk Hotel, Topeka, Kansas

Monday Morning, May 3

FLORENTINE ROOM

GEORGE E. BURKET, JR., M.D., Kingman, *Presiding*

9:30 Business Session

Monday Afternoon, May 3

FLORENTINE ROOM

SCIENTIFIC SESSION

GEORGE E. BURKET, JR., M.D., Kingman, *Presiding*

1:30 **Bernard A. Watson, M.D., Clifton Springs**
The Management of Obesity in an Office Practice

2:00 **James R. Cook, M.D., Cleveland**
Hormone Therapy of Cancer

2:30 Intermission

2:45 **Bernard A. Watson, M.D., Clifton Springs**
The Hypometabolic State as the Cause of Fatigue

3:15 Panel Discussion—Questions and Answers

Monday Evening, May 3

ROOF GARDEN

GEORGE E. BURKET, JR., M.D., Kingman, *Presiding*

6:30 Symposium

7:30 Banquet

THE HON. WALTER H. JUDD, Congressman from Minnesota, *Guest Speaker*
Dancing to Music of George Tidona

REFERENCE COMMITTEES

Dr. Lucien R. Pyle, president of the Kansas Medical Society, announces the appointment of two Reference Committees which will serve for the 1954 annual session. One committee will review all reports published in this issue of the JOURNAL. Another will review all new business. Both groups will meet at a specified time and place during the meeting. Any person having suggestions to make regarding business introduced to the House of Delegates should contact a member of the appropriate committee or appear before the committee in session.

Membership on the committees is as follows:

COMMITTEE ON REPORTS: Donald P. Trees, M.D., Wichita, Chairman; J. Philip Berger, M.D., Wichita; Leland F. Glaser, M.D., Hutchinson; Victor H. Hildyard, M.D., Baldwin; Bruce P. Meeker, M.D., Wichita; Earl L. Mills, M.D., Wichita; Richard O'Donnell, M.D., Ellsworth; L. W. Shepard, M.D., Larned; W. A. Smiley, Jr., M.D., Junction City.

COMMITTEE ON NEW BUSINESS: Lee H. Leger, M.D., Kansas City, Chairman; Clarence H. Benage, M.D., Pittsburg; J. Gordon Claypool, M.D., Howard; Francis T. Collins, M.D., Topeka; R. Dale Dickson, M.D., Topeka; Edward J. Ryan, M.D., Emporia; W. F. Roth, Jr., M.D., Kansas City; Robert Sohlberg, M.D., McPherson.

Annual Banquet

Wednesday, May 5, 1954, 7:00 p.m.

Topeka Country Club, 27th and Buchanan Streets

LUCIEN R. PYLE, M.D., Topeka, President, Kansas Medical Society, *Presiding*

PROGRAM

Pauline Deniston, Soprano, Star of 1953 Season, Kansas City Starlight Theatre

Vera McNary and her Strata Marimba, Popular Entertainer Seen on the Ed Sullivan "Toast of the Town" Television Show

Dancing to the Music of Johnny Coon and His Orchestra

The Shawnee County Medical Society takes special pleasure this year in being host to the annual meeting since, for the first time, the banquet will be held in a place noted for its attractiveness. The new club house of the Topeka Country Club, with adequate parking space, is one of the most beautiful buildings in the state, and its decor and cuisine have been the subjects of many published articles.

The price of the banquet ticket, $6.00, covers all costs—the banquet, the dance, and individual refreshments, including ice—throughout the evening. Performers of exceptional ability will be presented during the banquet program. There will be no speakers. The entire evening will be devoted to entertainment and enjoyment.

Attend the Annual Banquet **The Big Social Event of the Meeting**

Kansas Chapter, American College of Chest Physicians

Annual Meeting, Tuesday, May 4, 1954

Topeka, Kansas, Kansan Hotel, Walnut Room

Non-Members Invited

4:00 X-ray Conference (Panel Discussion)

C. F. Taylor, M.D., Norton, Moderator

Homer L. Hiebert, M.D., Topeka, Radiologist

Martin J. FitzPatrick, M.D., Kansas City, Chair of Chest Diseases, University of Kansas Medical Center

Arthur L. Ashmore, M.D., Wichita, Chest Surgeon

Each member of the College will present two cases with brief histories and x-ray pictures. Non-members may also present cases.

6:00 Social Hour

7:00 Dinner

Executive Board Meeting

Kansas Association of Clinic Managers

May 3 and 4, 1954

Topeka, Kansas, Kansan Hotel

Monday, May 3

7:00 Social Hour

Tuesday, May 4

KANSAN ROOM

9:00-4:00 Business Session and Program

Collection Techniques—Arthur Holtman, Nelson Clinic, Manhattan

Health and Accident Insurance—Don Taylor, Gelvin-Haughey Clinic, Concordia

Health and Accident Insurance—Walter Good, Hertzler Clinic, Halstead

Kansas Medical Assistants' Society

Annual Meeting, May 2 and 3, 1954
Topeka, Kansas, Kansan Hotel

Sunday, May 2

9:00 Registration and Coffee
Mezzanine

AFTERNOON SESSION

ROOF

2:00 Francis T. Collins, M.D., Topeka, President, Shawnee County Medical Society

Address of Welcome

2:15 Lucien R. Pyle, M.D., Topeka, President, Kansas Medical Society

Response to Address of Welcome

2:30 Panel on Medico-Legal Problems

Harry J. Davis, M.D., Topeka
The Physician's Viewpoint

Mr. Barton E. Griffith, Topeka
Legal Aspects

Mr. John M. Schroeder, Topeka
Insurance and Workmen's Compensation

3:30 Question and Answer Session on Medico-Legal Problems

4:00 Business Session and Election of Officers

6:30 Dinner and Entertainment—"Then and Now"

Monday, May 3

9:00 Registration
Mezzanine

MORNING SESSION

ROOF

10:00 Mrs. Lerah Lacey, Topeka, President, Kansas Medical Assistants' Society

Meeting Called to Order and Announcements

10:10 Miss Fern Schaake, Topeka, President, Shawnee County Medical Assistants' Society

Greetings

10:15 Mr. Herbert R. Allee, Kansas City, Missouri, Assistant to Branch Manager, Parke, Davis and Company

Research and Development of Therapeutic Agents

10:45 Mrs. Margaret Foster, Topeka, Kansas Blue Shield

What the Medical Assistant Should Know about Blue Shield

Question and Answer Period on Blue Shield

11:15 Lawrence Bee, Ph.D., University of Kansas, Lawrence

Marriage Is Not Made in Heaven

12:30 Luncheon and Program
Mrs. George P. Bishop, Topeka

A Volunteer in a Mental Hospital

AFTERNOON SESSION

2:30 Installation of Officers

3:00 Viewing of Scientific and Technical Exhibits

Municipal Auditorium, South Entrance

Woman's Auxiliary to the Kansas Medical Society

Annual Meeting, May 3, 4, 5, 6, 1954

Topeka, Kansas

Monday, May 3

1:00-4:00 Registration

Municipal Auditorium, South Entrance

Tuesday, May 4

9:00-4:00 Registration

Municipal Auditorium, South Entrance

10:00-Noon Welcoming Coffee

Home of Dr. and Mrs. William H. Crouch, Jr., 2504 Granthurst

Transportation Provided

12:30 Past State Presidents' Luncheon

Elks Club, Seventh and Jackson

2:00 Pre-Convention Board of Directors Meeting

Municipal Auditorium, Room 102-A

6:30 Dinner Party and Fashion Show

Jayhawk Hotel, Roof

Wednesday, May 5

9:00-4:00 Registration

Municipal Auditorium, South Entrance

9:00 General Session

Municipal Auditorium, Room 102-A

1:00 Luncheon Honoring Mrs. Leo J. Schaefer, National President, and New State Officers

Hotel Jayhawk, Roof

3:00 Post-Convention Board of Directors Meeting

Hotel Jayhawk

7:00 Annual Kansas Medical Society Banquet

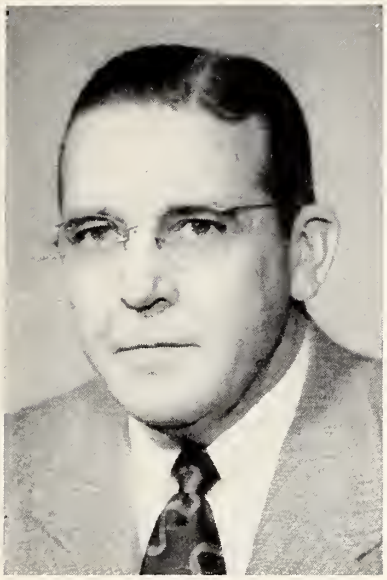
Topeka Country Club, 27th and Buchanan

Thursday, May 6

9:00-Noon Registration

Municipal Auditorium, South Entrance

President



Lucien R. Pyle, M.D., *President*

Lucien R. Pyle, M.D., of Topeka, will conclude his presidency of the Kansas Medical Society at the close of this annual session. One year ago the JOURNAL reflected upon his distinctive qualities of leadership and predicted he would guide the affairs of this Society with integrity and courage toward the practical fulfillment of its highest ideals. That his accomplishments have not only borne out this prophecy but exceeded even these high expectations will be acknowledged by all who have interested themselves in the affairs of their Society.

His might well be remembered as the year of decision, a period of precarious balance where a single impetuous action could have turned the Society into fateful directions. Not only once but repeatedly did this situation confront him, and without exception his calm mature wisdom, his unfaltering idealism, and his uncompromising honesty prevailed. Many examples could be selected to illustrate this statement, among which the following is just one. While it will quite possibly be some years before the final solution to the indigent care problem is achieved, the formula has been prepared. The beginning decision has been made, so there remains only the need to pursue the course to its gradual conclusion. Nor was this the only problem. In this office problems come daily, and Dr. Pyle gave each, the large and the small, a thoroughly studied opinion.

He personally visited almost every county medical society in this state. He attended numerous committee sessions and has often represented the Society in meetings of allied professional and other groups. Wherever he has been, he inspired the confidence of those he met. His presidency has added to the prestige of medicine in Kansas and to its dignity. The Society will remain in his debt and, with sincerest gratitude for his unselfish and magnificent leadership, will recall this among its greatest years.

President-Elect

No thinking person can deny that this year might well become the one most perilous period in the nearly 100-year history of this Society. This somber forecast, while endorsed by your incoming president, is infinitely softened by the personality, the character, and the ability of the man elected to the office of highest responsibility within the Society. Murray C. Eddy, M.D., of Hays, is most acutely aware of the problems awaiting his presidency, but the proven abilities, the experience, and the inspiration he will bring to this office leave the outcome assured of success.



Murray C. Eddy, M.D., *President-Elect*

Problems of federal legislation are visible upon the horizon which might well grow into a magnitude never before experienced in medical history. Situations within medicine itself are delicately poised, awaiting only the persuasion of a voice with conviction to guide a future course. In Kansas a myriad of herculean tasks is at hand. Dr. Pyle gave a great many of these problems a direction, but strong, sure leadership is now essential to chart a steadfast future course—and much of it into the unknown.

All this falls to the responsibility of your incoming president, who is most splendidly qualified to fulfill his task. His familiarity with the work of the Society, his long years of service, his presence on the Board of Medical Registration and Examination assure his experience. His success as a physician assures his ability. But most of all, his genial enthusiasm, that indefinable talent reflected in his personality, assures his unique qualities of leadership which will guide the Society through a glorious year.

PRESIDENT'S PAGE

DEAR DOCTOR:

In writing this, my last page as your president, there are a few remarks and observations that I would like to make as a matter of public record.

I continue to be amazed at the immense amount of time, thought, and financial outlay that so many of you doctors give so willingly each year in order to equitably solve the problems of the Kansas Medical Society in its endeavor to furnish the highest possible type of health care to the people of Kansas. I am sure that each one of us would be greatly surprised at the aggregate of those hours and the total amount of individual expense. And all of this is in addition to the time you spend in county medical societies and hospital staff organizations.

It is my most considered thinking that whatever success has been attained by the Kansas Medical Society during the past year has been brought about through the earnest effort, the clear thinking, and the hard work of the membership of our committees. To my certain knowledge there has been no committee that has fallen down on the job. Some of the committees, by virtue of their constitutional purpose, have been relatively inactive. But the remaining committees have been extremely active, not only in carrying on the projects held over from last year but also in formulating and working on new projects.

Your councilors and officers have been most attentive, prompt, and straight forward in the performance of their duties. There have been two long and arduous Council meetings as well as considerable demand upon their resources in settling issues that were sent to them through the mail.

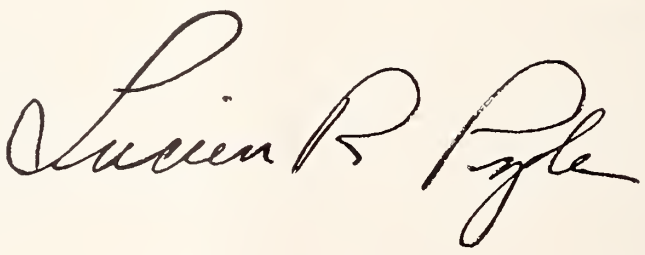
It is by virtue of these efforts that the Kansas Medical Society cannot and will not fail to live up to the statement made in its original charter of "perpetual succession forever."

One cannot be president of the Society for a year without realizing the true worth of our executive secretaries. Their conscientious devotion to us and our profession is marvelous indeed.

As I close this year, I need not ask you to continue the same fine work during the coming year, because I know that you will. I also know that with each advancing month you will realize that your selection of Murray Eddy as your president was both wise and fortunate.

I want to thank each and every one of you for your fine spirit, your help, and your co-operation. I am deeply appreciative of this great honor that you have given me. It has been a pleasure to be your president, and I have enjoyed every minute of it. I sincerely hope that I have satisfactorily fulfilled the trust that you placed in me.

Thank you, best wishes, and "perpetual succession forever."



Councilor Reports

First District

To the House of Delegates:

The past year has been rather quiet. There have been a few minor difficulties which, thanks to the individual members, were settled on a local basis.

One district meeting was held at Centralia with a nice attendance. Dr. L. R. Pyle and Mrs. I. J. Waxse honored us with their presence.

Due to the military demands, fewer new men have set up practice in the district. There are still a number of locations which need medical coverage.

This being my last year of service as councilor, I want to express my appreciation of the wonderful help and courtesy that have been accorded me by everyone in the past six years. It has been a pleasure to have held my office.

Respectfully submitted,

W. L. Anderson, M.D., *Councilor*

Second District

To the House of Delegates:

An analysis of activities in the second district indicates no startling problems to have originated during the past year. Your councilor has visited several of the societies within this district and has generally found interest in medical affairs to be high. In fact, comments concerning the medical situation in this district would generally parallel those experienced in other areas. It appears unnecessary to record them at this time.

We do, however, have two distinctive situations, the first of which is in Linn County. Several years ago, all physicians in Linn County withdrew from the Kansas Medical Society and left that county alone in this state without representation in the Kansas Medical Society. Your councilor has met with the physicians in Linn County and reported his findings during a meeting of the Council. When it appeared that these doctors wished to reunite with the Society, the Council agreed to issue this county a new charter and relieve each member from the responsibility of the payment of back dues. This was reported, but to date there has been no further interest shown by the involved physicians. That situation, therefore, has not been solved.

The second situation which might be worthy of comment in such a report is that several communities within this district which have long been without hospital facilities, or at least without adequate hospitals, are now building hospitals. Among these are Burlington and Olathe, which opened their hospitals about the middle of March. It is our opinion that this will offer a considerable improvement in the total health picture of this one remaining area of the state that lacked adequate hospital facilities and we are pleased to report that fact as a significant advancement within this district.

Respectfully submitted,

H. Penfield Jones, M.D., *Councilor*

Third District

To the House of Delegates:

I wish to report that no serious problems have arisen within the societies composing the Third District during the last year.

There is not an acute shortage of physicians in the Third District. Several new hospitals have been constructed or are in the process of being constructed in the district during the past year.

The Southeast Kansas Medical Society, which is made up of the component societies of the Third District, has been quite active.

Respectfully submitted,

Charles E. Vestle, M.D., *Councilor*

Fourth District

To the House of Delegates:

Physicians in the Fourth District have enjoyed a pleasant professional relationship during the past year. Most of the men called to military service have returned to practice. Hospital facilities in the district were greatly improved with completion of the addition to Stormont-Vail Hospital in Topeka, adding from 75 to 90 beds to those already available.

The councilor from this district can offer one criticism at present—the membership's seeming lack of interest in Society affairs. Early in the year the Shawnee County Society made plans for a meeting to which physicians in nearby county societies were invited, but the session was later cancelled because of poor response.

It is hoped that this situation will be improved during the coming year, when a legislative session will be held. The co-operation of all physicians is especially important in legislative years, and the best interests of medicine are served when each individual physician keeps himself informed of medical problems and assists in their solution.

Your councilor has attended all meetings of the

Council during the past year and has enjoyed this participation in Society affairs.

Respectfully submitted,

FLOYD C. TAGGART, M.D., *Councilor*

FIFTH DISTRICT

To the House of Delegates:

Good will, friendly, social and professional cooperation and gradual but distinct raising of the levels of efficiency in medical care characterize the membership of the county societies of this district.

Three of our societies have tri-county meetings each year, alternating between McPherson, Marion, and Newton, a plan I commend to others of the smaller societies of the state.

The highlight in our district is the prospect of the meeting of the Kansas Medical Society in Hutchinson in 1955. Neighboring societies will co-operate with the Reno County Society to help make this meeting a complete success.

Respectfully submitted,

H. M. GLOVER, M.D., *Councilor*

SIXTH DISTRICT

To the House of Delegates:

Your present councilor is serving on an interim appointment basis. During this short tenure there have been no major problems. No visits have been made to the societies within the district because of the limitation of time, and no requests have been received from any of the county societies involved. The activities of this district appear to have followed a normal course during this period.

The Sedgwick County Medical Society has commenced construction of its own building consisting of executive offices, library, and auditorium. Completion is expected in the fall.

Respectfully submitted,

NORTON L. FRANCIS, M.D., *Councilor*

SEVENTH DISTRICT

To the House of Delegates:

There has been no particular medical activity in this district the past year. One or two minor local problems have arisen that were well handled by the local society.

There has been little, if any, comment concerning the rezoning of councilor districts. From this it is presumed that the problem can be more easily settled at the state level.

Some mild effort has again been made to consolidate two or three societies in an attempt to have better society meetings, but no society seems willing to give up its identity.

Respectfully submitted,

S. A. ANDERSON, M.D., *Councilor*

EIGHTH DISTRICT

To the House of Delegates:

Medical activities in this district have followed a normal pattern in the last year. There have been no particular problems. Some new doctors have located in the district, and hospital facilities have been improved. We feel that medical services are adequate and in general operating smoothly.

Conditions in general are quite good too. The drought hasn't affected this area particularly so far. Business activity may be off some, but is still at a high level.

Respectfully submitted,

ANDREW E. RUEB, M.D., *Councilor*

NINTH DISTRICT

To the House of Delegates:

Since the last councilor's report, there have been a few changes in the Ninth District.

The new hospital at Hill City has been opened and dedicated. Dr. O. Dale Smith has left Hill City for a residency in radiology.

Dr. B. Dan Ferguson, formerly of Bird City, is now serving in the Army. Dr. Findley Law of Kansas City has moved to Bird City, where he is now in practice.

Dr. Ward Benkleman, who has been associated with Dr. J. H. A. Peck in St. Francis for several years, has moved to Polson, Montana.

Dr. Kenneth Knuth, absent from Atwood for naval service, is expected to return in April and will be associated with Dr. C. E. Henneberger.

Dr. Harry R. Custer, formerly with the Eddy Clinic in Hays, is now located in Colby.

The circuit courses being held at the Country Club in Colby have been good and well attended.

Your councilor has attended the two Council meetings, called by the president, Dr. Lucien R. Pyle, in Topeka.

Respectfully submitted,

JAMES L. JENSON, M.D., *Councilor*

TENTH DISTRICT

To the House of Delegates:

The Tenth District has had no great problems or difficulties in the past year. With the exception of

three men still in military service, all those who were recalled have returned to practice.

The physicians in the district meet quarterly as the Central Kansas Medical Society, and we have both scientific and business meetings at this time. Also the circuit course of the University of Kansas School of Medicine, held at Hays this year, has given us ample opportunity to take care of district business.

Your councilor has attended each of the called meetings of the Council and has enjoyed being the representative of the Tenth District.

Respectfully submitted,
L. W. Reynolds, M.D., *Councilor*

Eleventh District

To the House of Delegates:

There has been very little activity at the local level in this district. The doctors were requested to contribute to the American Medical Education Foundation, and a number of them did so. The members of the Pratt County Medical Society came through 100 per cent.

It is hoped that the remaining doctors of this district will get interested in this project which can be a big booster to the University of Kansas School of Medicine.

Respectfully submitted,
Cyril V. Black, M.D., *Councilor*

Twelfth District

To the House of Delegates:

There has been more medical activity in the Twelfth District in the past year. One district meeting was held, and societies in Seward and Finney counties function regularly in conjunction with hospital staff meetings. The Ford County society is now having quarterly scientific meetings, in addition to the regular monthly business meeting following each staff meeting.

The University of Kansas School of Medicine postgraduate extension courses have been held in Dodge City this year with a large attendance at each session.

Medical service has improved in the Twelfth District with adequate physicians and new rural hospitals.

Respectfully submitted,
R. G. Klein, M.D., *Councilor*

Kansas Blue Shield

To the House of Delegates:

The year 1953 was very much like 1952 for Blue Shield. Both were good years in many ways. Here are several high points of the year just past:

Enrollment. Net enrollment increased 41,403 members as compared to 30,221 members in 1952. (Gross new enrollment was over 90,000 members in 1953.) The cancellation rate was lower than at any previous time in the plan's history. The extended benefit rider (covering polio and 8 other dread diseases) was offered for the first time and accepted by over half our members. It produced a significant contribution to reserve, and we have every reason to feel it met a need.

A new non-group enrollment program has been developed. This new program includes most of the safeguards usually considered necessary for non-group enrollment. First presented to the public in late January 1954, the plan had received 3,713 applications by March 8, indicating another excellent response to a new offering. (These figures do not include Shawnee and Sedgwick Counties, which opened on a continuous basis March 22. No reports are yet available on these two experimental counties.) The staff of Blue Shield is meeting with county medical societies as rapidly as possible, explaining this new program, including the use of the new health statement which has to be completed by the applicants.

New Headquarters Building. The year 1953 saw our board act on the housing needs of the organization. Together with the Blue Cross Board they decided a new building was necessary for the continued economical operations of the staff. Jointly they authorized construction in Topeka of a modern-style office building to cost approximately $450,000 with land. Cost of space in our new building will be substantially less than the space used in our present rented quarters. Construction began in October, and the contractor estimates the completion date will be this coming October, just one year later.

Finances. Blue Shield has a plan reserve of a little over $1,150,000. About 8.7 per cent of the income was placed in reserve in 1953. The reserve as of the end of 1953 equalled about 4.1 average months of case and operating expense. In general terms, Blue Shield plans throughout the United States have set up, as a criterion, about 5 months of case and operating expense as a safe reserve. We expect to reach this

position during 1954. From the financial standpoint alone, it appears that the present rates should be adequate for the present benefits until some new increasing trend in the use of the services starts developing. All indications at the moment point to the fact that utilization of hospital and physicians' services has leveled during the last half of 1953. The board will give careful consideration to the recommendations of the Committee on Blue Shield Fee Schedule, which appear elsewhere in this issue.

Income and Payments. The year 1953 made history for Blue Shield in still another way. For the first year since the plan's beginning, the total number of cases serviced (both major and minor) passed the 100,000 mark. (Exact figure 100,763.) These services represent a total payment to physicians of $2,901,096.17 for the year. Since total plan income was $3,677,-101.38, this indicates a continuing level of usage around 80 per cent.

Operating Expenses. Operating the Blue Shield plan takes in such activities as enrolling new members, collecting and accounting for their dues, and processing cases for payment. To do this for Blue Shield's 336,509 members cost $457,153 during 1953. This amounted to 12.46 per cent of income from dues compared to 11.8 per cent of income from dues during 1952. This slight increase primarily resulted from the cost of offering and administering the extended benefit rider, and from the necessary expansion of our field staff to produce greater enrollment during 1954.

While 1953 has been notable for Kansas Blue Shield, the national effort continues equally promising. Membership in all Blue Shield plans reached 28 million by the end of 1953. Despite such growth, both in Kansas and out, our Blue Cross and Blue Shield plans are actually enrolling a gradually declining share of the national "market" for health protection. Competition is particularly keen. The larger insurance companies are making a strong bid for the "best risk" groups. We could wish that the better companies might also take on some of the community responsibility which is being carried by Blue Cross-Blue Shield. This community responsibility, which Blue Cross-Blue Shield accepts, is the strongest reason why Blue Shield participating physicians should give extra support to Blue Cross-Blue Shield.

Respectfully submitted,

HENRY S. BLAKE, M.D., *President*

Kansas State Advisory Committee

To the House of Delegates:

In June 1953, Dr. B. J. Ashley was appointed chairman of the Kansas Advisory Committee upon the resignation of Dr. Lucien R. Pyle to accept the presidency of the Kansas Medical Society.

There has been little activity since August of 1953 when the last call for doctors was issued. At that time the armed forces requested Selective Service to stop processing until further notice, and until the present there has been no call for additional doctors. It is expected that the resumption of drafting doctors may begin in July 1954, but no definite plans have been released as yet.

There have been 95 doctors from Kansas commissioned into service to date. Of these, 61 are still in service, 4 have been discharged, and 29 are in the reserves.

Kansas has had calls for 73 doctors for induction. All of these have accepted a commission in lieu of induction except one, who was inducted, then later discharged, and accepted a commission. There are approximately 56 doctors in Kansas in Class II-A who may expect to be called if needed.

It may be of interest if certain factors of the so-called Doctor Draft Law were reviewed at this time. The two most important changes in the amendment of June 29, 1953, were as follows: First, it added two new classifications, Class II-S, students, and Class IV-A, registrants who have had more than 21 months active duty in the armed forces. The second change was in length of service for Priority II registrants from 21 months to 17 months active duty.

The four priorities under the Doctor Draft are as follows: Priority I—those given government assistance or deferred during World War II to pursue specialized training and who have had less than 90 days active duty in the armed forces.

Priority II—same as above except that they have had 90 days or more but less than 17 months' active duty in the Armed Forces.

Priority III—no active service subsequent to September 16, 1940.

Priority IV—those not in first or second priority who have had active service subsequent to September 16, 1940.

There is a new uniform policy applicable to both reserves and special draft registrants. Hospitals are cautioned not to plan on residencies for men in Pri-

orities I and II. The directive states: "Practically without exception these men should be considered available for service and should not be deferred to accept or to continue in residencies for another hospital year. If they desire to serve the hospital temporarily until such time as they are called, there is no objection, providing it is understood they are not to be delayed beyond the time that they are called into service."

Priority III men with obligations under the regular draft, and who were under 29 years of age last June 19, "should enter service on completion of their internship without exception," the directive declares. It points out, however, that, if they join the reserves, they will be called to service "in an orderly manner." Priority III registrants between 28 and 31 years of age also "should enter service as soon as they have completed their internship or current residency." The objective is to get the young non-veterans of the first three categories processed into the reserves, in order that the induction age can be kept at 31 and the use of the Doctor Draft avoided for the next year.

The various advisory committees have been very prompt and helpful in all phases of the Kansas Advisory Committee's operation and in keeping the files up to date.

Respectfully submitted,

B. J. Ashley, M.D., *Chairman*

Committee Reports

Allied Groups

G. E. Kassebaum, El Dorado, Chr.; C. H. Benage, Pittsburg; P. S. Combs, Leavenworth; D. R. Davis, Olathe; R. D. Dickson, Topeka; A. C. Dingus, Yates Center; E. E. Henderson, Columbus; Harry Lutz, Augusta; R. R. Snook, McLouth.

To the House of Delegates:

This committee had a meeting at which time a considerable discussion pertained to the Interprofessional Advisory Council. One year ago this committee reported that the formation of such a council virtually eliminated the need for continued existence of this committee. On this occasion, however, the purpose of the council was clarified to indicate that agreements and policies may be recommended by the council but that each member agency must provide some means for implementing those recommendations. This committee volunteers its services in that behalf and will await recommendations from the Interprofessional Advisory Council.

There are several recommendations made by the committee which require approval of the House of Delegates. It was felt that an annual course in first aid for undertakers and ambulance drivers might well be given under the auspices of the University of Kansas School of Medicine and that Civilian Defense might aid in defraying the cost of such education. If the House of Delegates will approve this recommendation, the committee will proceed further with the project.

The second was the recommendation from the committee that doctors of dental surgery be generally admitted to meet with hospital professional staffs and that such individual doctors of dental surgery as appear to be qualified within their field should be welcomed to perform oral surgery within the hospital. Again, this recommendation is subject to approval of the House of Delegates.

Two other items were recommended for further committee effort. The physicians of Kansas should interest themselves more actively in nurse recruitment programs, and, finally, the committee should meet with the life and the health and accident underwriters of this state.

Respectfully submitted,

G. E. Kassebaum, M.D., *Chairman*

Anesthesiology

E. M. Sutton, Salina, Chr.; L. L. Bresette, Kansas City; H. J. Brown, Winfield; P. H. Lorhan, Kansas City; R. S. McKee, Leavenworth; C. D. McKeown, Wichita; R. T. Parmley, Wichita; F. C. Taggart, Topeka.

To the House of Delegates:

This committee held one meeting during the past year. The group reviewed suggested projects and discussed sponsoring graduate courses in anesthesiology for Kansas physicians. They decided that the yearly postgraduate program at the University of Kansas School of Medicine is a successful venture, as evidenced by the increasing enrollment and national recognition. Courses are arranged to suit the needs of the individual physician.

A discussion in regard to civil defense led to few conclusions but many questions in regard to anesthesiology in case of emergency. The Civil Defense Committee does not have an anesthesiologist on its

roster, and the following questions were asked of that committee. In case of catastrophe, who is going to give anesthetics to the injured who require surgery? Has any consideration been given to the stockpiling of the various anesthetics? With a paucity of anesthesiologists in the state, what method of supervision will be utilized for the instruction of physicians in the use of anesthesia? Who will be responsible for the blood and fluid replacement program? Has consideration been given to the management of patients who have respiratory problems? Who shall decide what anesthetics are best for the patient?

A list of guest speakers on anesthesia was sent to the Executive Office for consideration by the program committee for the annual session.

The committee is interested in establishing a mortality study commission. All fatalities occurring within the first 24 to 48 hours after surgery would be reported to the commission on approved forms prepared by the Kansas Medical Society. The reports, of course, would be anonymous. Such a commission would require the wholehearted support of the medical society, and a nominal budget for expenditures would be needed. The commission should be comprised of an anesthesiologist, a surgeon, an internist, a pathologist, and probably a physiologist or a cardiologist. It should be large enough to render an authoritative opinion but small enough to function smoothly and rapidly. In regard to the decision the commission should render, the committee believes it should be one of two, namely preventable or unpreventable. A decision that a death was unpreventable would close a case. The committee recommends that such reports be published in the JOURNAL. The committee will continue the study of this problem.

Respectfully submitted,
E. M. SUTTON, M.D., *Chairman*

AUXILIARY

I. J. Waxse, Oswego, Chr.; L. J. Schaefer, Salina, Vice Chr.; F. T. Collins, Topeka; C. O. West, Kansas City.

To the House of Delegates:

With one exception, all members of the committee were present at a meeting held at Salina, February 3.

It was agreed that the magazine *Today's Health* should be more widely read by the Society, and ways of bringing that about were discussed.

A change in the postal handling of the *Auxiliary News* was discussed and rejected.

Something new has been created this year by the auxiliary in the establishment of a memorial fund wherein, instead of bestowing flowers to the memory of deceased esteemed friends, the auxiliary will accept contributions to a nurse scholarship fund. Contributions may be sent to Mrs. William Brewer of Hays.

It was the consensus of this committee that the auxiliary should stay in certain fields in its program and not try to branch out too far. It was felt that they could do a better job if they concentrated on such projects as organization, *Today's Health,* and public relations.

The Society should be honored to have a Kansas woman as head of the Woman's Auxiliary to the American Medical Association this year. We refer to Mrs. Leo J. Schaefer, Salina, who is doing a fine job as head of this group.

The committee feels that much benefit would be derived from a closer co-operation between the Society and the auxiliary. The doctor's wife is a constant and willing worker and should be brought into closer co-operation with the aims and plans of the Society.

Respectfully submitted,
I. JOSEPH WAXSE, M.D., *Chairman*

BLUE SHIELD FEE SCHEDULE

N. L. Francis, Wichita, Chr., ENT; H. O. Anderson, Wichita, Orthopedics; C. M. Barnes, Seneca, General Practice; W. L. Beller, Topeka, Radiology; P. D. Brown, Salina, Obstetrics and Gynecology; G. F. Gsell, Wichita, Eye; A. G. Isaac, Newton, Urology; B. I. Krehbiel, Topeka, Pediatrics; W. O. Martin, Topeka, Anesthesiology; E. J. Ryan, Emporia, Internal Medicine; L. L. Saylor, Topeka, Surgery; B. E. Stofer, Wichita, Pathology; G. G. Whitley, Douglass, Blue Shield.

To the House of Delegates:

Your committee has held two meetings during the year. At the first, results of various surveys of fees were evaluated. As a result of these surveys, recommendations in the fee schedule were made. A discussion of fees associated with non-surgical cases ensued. As a result of this, a subcommittee, with Dr. E. J. Ryan of Emporia as chairman, was appointed to study this problem and report back to the next meeting of the committee. Various letters from participating physicians concerning fees allowed were read, and disposition was made in the most equitable manner at this time.

At the second meeting, the committee went on record as recognizing the problems of fees associated with non-surgical care and the equality as associated with surgical fees. The report of Dr. E. J. Ryan's subcommittee was received. On the basis of this report, further recommendations were made to the Blue Shield Executive Board concerning non-surgical care. It was further recommended that another subcomit-

to be appointed to study and report on specific problems related to psychiatry.

It is the hope of this committee that as a result of its activity, closer harmony with all physicians in the state can be obtained.

Respectfully submitted,

NORTON L. FRANCIS, M.D., *Chairman*

BLUE SHIELD RELATIONS

L. F. Glaser, Hutchinson, Chr.; J. O. Austin, Garden City; E. S. Brinton, Wichita; O. R. Cram, Larned; A. J. Horejsi, Ellsworth; C. S. Joss, Topeka; M. D. McComas, Jr., Concordia; D. E. McCoy, Oberlin; G. E. Manahan, Lawrence; J. E. Seitz, Wakeeney; C. E. Stevenson, Neodesha; R. M. Thomas, Marysville.

To the House of Delegates:

Two meetings of the full membership of the Blue Shield Relations Committee have been held during the past year. At the annual meeting, the year's program was outlined. At the midyear meeting held in Hutchinson, recommendations of the Blue Shield Executive Committee were discussed. At that time a rate increase was contemplated by Blue Cross-Blue Shield, and some changes were recommended and approved in the Blue Shield Membership Agreement. Since the rate increase proved to be unnecessary, the contemplated changes were not made.

District meetings have not been held during the past year except in the four districts where elections to the Blue Shield Board of Trustees were necessary. It is now the duty of the Blue Shield Relations Committee to elect members of the Blue Shield Board of Trustees from nominations submitted by the county medical societies in the proper districts.

When the new program of community enrollment was launched shortly after the first of this year, members of the Physician Relations Committee co-operated with the staff by securing appointments for staff members to speak at county medical society meetings. This approach was considered better than holding District Physician Relations Committee meetings. During the coming year the district meetings may be reached if the program can be developed on that basis.

Respectfully submitted,

LELAND F. GLASER, M.D., *Chairman*

CHILD WELFARE

W. H. Crouch, Topeka, Chr.; M. S. Boyden, Lawrence; D. R. Davis, Emporia; H. P. Jubelt, Manhattan; G. M. Martin, Topeka; F. L. Menehan, Wichita; H. C. Miller, Kansas City; E. G. Padfield, Salina; L. N. Speer, Kansas City.

To the House of Delegates:

During the past year this committee has had five meetings. The first was held at the time of the 1953 annual meeting, at which time it was decided that the committee would take an active part in organizing a Kansas State Pediatrics Society. A motion approved at that meeting was to have child accident prevention as the chief project for the year.

The next meeting was held in the office of Dr. T. R. Hood, Kansas State Board of Health. The entire meeting was devoted to a study of the distribution of gamma globulin for the year 1953. The committee's recommendations were approved, and the membership was informed.

At the third meeting the committee discussed the newly organized Kansas State Pediatrics Society. It was also announced at this meeting that the specialty of pediatrics is now represented on the Blue Shield Fee Schedule Committee.

Considerable time was spent on an accident prevention program which is now in progress. Articles in the JOURNAL, letters to physicians, and an exhibit for the annual meeting of the Kansas Medical Society were among the suggestions accepted by the committee.

An outline entitled "Diagnostic Criteria for Poliomyelitis" was prepared and sent to the membership.

The fourth meeting was called by the National Foundation for Infantile Paralysis. This committee held a separate meeting following a lengthy discussion with the foundation and decided that the procedure outlined for the conduct of the experiment on the new immunization program for poliomyelitis was within the realm of scientific expediency and that safeguards for the individual patient were adequately provided. It approving this program, the committee requested that this action be called to the attention of the Council for its immediate action and further suggested that a letter requesting endorsement be sent to each county medical society in the state. This was done, and a report was received from almost all county societies.

The final meeting was held in Emporia with the Kansas State Board of Health, at which time the committee was asked for recommendations in regard to communicable diseases.

Respectfully submitted,

W. H. CROUCH, M.D., *Chairman*

CONSERVATION OF EYESIGHT

D. O. Howard, Wichita, Chr.; B. J. Ashley, Topeka; F. N. Bosilevac, Kansas City; H. L. Bryant, Coffeyville; L. L. Calkins, Kansas City; R. E. Cheney,

Salina; D. T. Loy, Great Bend; H. E. Morgan, Newton; W. M. Scales, Hutchinson; D. P. Trimble, Emporia.

To the House of Delegates:

This committee had several meetings during the past year and continued, as in the past, to co-operate with the Division of Services for the Blind of the Kansas State Board of Social Welfare. Upon this occasion members of that division appeared before the committee to report that a considerable increase in services had occurred during the past year and that a younger age group of patients was being cared for.

There was also a report on the newly created rehabilitation center at Topeka which currently serves not only Kansans but has received patients from 11 other states. The committee placed an official request with the board of social welfare for an increase in fees for a few selected services, especially those performed by opticians, but all were refused.

This committee has long recommended a restriction of the sale of fireworks and air rifles. Last year again the committee enlisted the aid of the Kansas Parent-Teacher Association in this effort. Together, and with interest from other organizations, the subject finally appeared before the state legislature, and some fireworks restrictions were placed into the statutes. Even though the present law is not strict enough, a beginning has been made, and the committee takes pleasure in noting that a considerable reduction in eye injuries resulting from fireworks occurred during the last Independence Day celebration. A poll taken among committee members revealed only one such accident occurring last summer.

It should also be noted that federal requirements governing the use of appropriations to the Division of Services for the Blind provided that optometrists must be used in state programs within the limit of their practice rights. A meeting was held this year with a committee from the Kansas Optometric Association and a formula agreed upon whereby optometrists may make examinations in Kansas but will not be entitled to render treatment. This formula has worked successfully up to the present time.

Respectfully submitted,

D. O. Howard, M.D., *Chairman*

Conservation of Hearing and Speech

LaVerne B. Spake, Kansas City, Chr.; C. W. Armstrong, Salina; E. L. Gann, Emporia; Lucien Gray, Wichita; J. A. Johnson, El Dorado; C. R. Kempthorne, Manhattan; W. D. Pittman, Pratt; G. O. Proud, Kansas City; M. J. Rucker, Sabetha; M. J. Ryan, Kansas City.

To the House of Delegates:

This committee, while not holding a formal meeting during the year, has continued to work on projects previously begun. These include clinics for children at the medical center as well as at Olathe and orientation classes for parents of deaf children.

Your chairman believes that a gradual increase in the number of hearing examinations for school children indicates that the effort of this committee during the past year is currently being recognized.

Respectfully submitted,

L. B. Spake, M.D., *Chairman*

Constitution and Rules

A. W. Fegtly, Wichita, Chr.; H. E. Haskins, Kingman; V. H. Hildyard, Baldwin; M. O. Steffen, Great Bend; C. E. Vestle, Humboldt.

To the House of Delegates:

In accordance with provisions of the constitution of the Kansas Medical Society, the Committee on Constitution and Rules now presents all amendments which have been suggested during the past year. These amendments will be discussed and voted upon at the meetings of the House of Delegates on May 4 and 6, 1954, in Topeka.

Numbers 1 and 2 relate to one subject:

1. Resolved: That the Constitution, Article VI, Section 1, be amended after the word "Society" by inserting a comma and the words "Chairman of the Editorial Board."

ARTICLE VI—COUNCIL

Section 1. The Council shall consist of one Councilor from each councilor district, and in addition the officers of the Society, CHAIRMAN OF THE EDITORIAL BOARD and the delegates to the American Medical Association as ex-officio members.

2. Resolved: That the Constitution, Article VII, Section 1, be amended after the word "councilors," by inserting the words "Chairman of the Editorial Board."

ARTICLE VII—HOUSE OF DELEGATES

Section 1. The House of Delegates shall be the primary legislative and governing body of this Society, and shall consist of the duly elected delegates, and the officers, councilors, CHAIRMAN OF THE EDITORIAL BOARD and past presidents of this Society.

COMMENT: Amendments No. 1 and No. 2 were read at the 1953 meeting and should be voted upon at the 1954 meeting. Their purpose, as explained last year, is to make the Chairman of the Editorial Board an official member of the Council and of the House of Delegates.

The committee recommends that resolutions 1 and 2 be adopted.

Numbers 3, 4, and 5 relate to one subject:

3. Resolved: That the Constitution, Article IV, Section 2, be amended by deleting the words "a second vice-president."

ARTICLE IV—COMPOSITION OF THIS SOCIETY

Section 2. The officers of this Society shall be a president, a president-elect, a first vice-president, [a second vice-president,] a secretary and a treasurer. All officers shall be elected by the House of Delegates of this Society for terms of office as are hereinafter provided.

4. Resolved: That the By-Laws, Chapter VI, Section 1, as amended in 1953, be further amended by deleting the words "First Vice-President," and by deleting the word "Two" and substituting therefore the word "THREE," and by deleting the word "Second" in the next line and substituting therefore the word "FIRST."

CHAPTER VI—ELECTION OF OFFICERS

Section 1. . . . the Committee shall meet not later than 90 days prior to the next annual session and present for publication in the Journal a list of candidates for each elective office, consisting of one or more candidates for the offices of President-Elect, [First Vice-President,] Secretary, Treasurer, and Delegate-Elect to the American Medical Association, and [two] THREE or more candidates for the offices of [Second] FIRST Vice-President and Alternate Delegate to the American Medical Association. . . .

The remainder of this section is not applicable to this amendment so is not reprinted here.

5. Resolved: That the By-Laws, Chapter VII, Section 4, be deleted and that the following sections be appropriately renumbered.

CHAPTER VII—DUTIES OF OFFICERS

[Section 4. The second vice-president shall also assist the president in the performance of his duties and in the event of the death, resignation, or removal of the first vice-president, shall succeed to that office for the remainder of the term.]

COMMENT: The Shawnee County delegation presented two resolutions at the 1953 session dealing with the officers of the Society and their election. Deleting the "Whereases" they were as follows:

(1). *Resolved* that the office of Second Vice-President be abolished, and *Resolved* that the vacancy on the Council, by abolishing the second vice-president, be filled by the Editor of the Journal.

(2). *Resolved* that a minimum of three names be offered as candidates for the office of First vice-president with a provision to permit nominations from the floor.

The first of these would require a constitutional amendment, and, being presented at one meeting, should be voted upon at the succeeding session. It would also require By-Laws changes. Since portions of these Shawnee County amendments are already incorporated in Constitutional amendments No. 1 and No. 2, we consider that amendments 3, 4 and 5, if passed, will make the wishes of the delegates from Shawnee prevail.

The Committee recommends that the office of second vice-president NOT be abolished, but favors the requirement that three or more candidates be nominated for the offices of second vice-president and Delegate-Elect to the American Medical Association. Therefore, the committee recommends that amendments 3 and 5 NOT pass and that amendment 4 be altered to include only the substitution of the word "three" for the word "two."

Numbers 6 and 7 relate to one subject:

6. Resolved: That the By-Laws, Chapter XII, Section 7 be amended by inserting a period after the word "Council" and deleting the remainder of that section.

CHAPTER XII—COMPONENT SOCIETIES

Section 7. Any physician who may feel aggrieved by the action of the component society of his county in refusing him membership, or in suspending or expelling him, shall have the right to appeal to the Council. [whose decision shall be final as to whether or not the appellant shall hold membership in this Society.]

COMMENT: Present wording of this section would appear to negate the time-honored right of each component society to be the absolute judge of its membership. The above deletion would still reserve the right of appeal to a censored or rejected member and would allow the Council to make recommendations to the component society.

7. Resolved: That the By-Laws, Chapter XII, Section 8 be amended by inserting after the word "compromise" the following: "Shall be the responsibility or duty of the Councilor of that district, and . . ."

CHAPTER XII—COMPONENT SOCIETIES

Section 8. In hearings of appeals, the Council may admit oral or written evidence as in its judgment will best and most fairly present the facts, but in every case of appeal efforts for conciliation and compromise SHALL BE THE RESPONSIBILITY OR DUTY OF

THE COUNCILOR OF THAT DISTRICT, AND shall precede all hearings.

COMMENT: This would emphasize the fact that efforts for conciliation or compromise should be made through the efforts of the councilor of the affected district.

The committee recommends the passage of amendments 6 and 7.

---

8. Resolved: That the By-Laws, Chapter XI, Section 7 be amended by inserting after the words "Executive Committee" the phrase, "who shall determine that the anticipated expenses of the meeting are within the budget."

CHAPTER XI—COMMITTEES

Section 7. The Committee on Arrangements . . . shall prepare and arrange for diversified scientific programs subject to the approval of the Executive Committee WHO SHALL DETERMINE THAT THE ANTICIPATED EXPENSES OF THE MEETING ARE WITHIN THE BUDGET and shall have general charge of all the annual session arrangements. . . .

The remainder of the section does not apply to this resolution.

COMMENT: This amendment was suggested in order that under no circumstances can the arrangements committee plan for and carry on a program for a meeting whose cost will be in excess of available funds of the Society.

The committee approves the adoption of this resolution.

Numbers 9 and 10 relate to one subject:

9. Resolved: That the By-Laws, Chapter IX, Section 1 be amended by deleting the entire section after the word "defense" in line 11 and substituting the words "To assist such member in planning for defense and IF REQUESTED by the defendant and deemed advisable by the Defense Board procure the services of an attorney approved by The Kansas Medical Society, provided the entire cost of such services shall be paid by the defendant. The Society shall not be obligated to pay ANY fees for legal services, nor any costs or judgments arising out of any action involving the professional services of any member."

CHAPTER IX—DEFENSE BOARD

Section 1. Members of The Kansas Medical Society are urged to carry individual professional insurance with commercial companies as these usually include possible indemnity in their policies, but additional assistance of the Society Defense Counsel may be provided if necessary or desired. It shall be the duties of the members of the Defense Board, severally or collectively, to investigate all claims of malpractice made against members desiring Society assistance; to assist in conducting the defense; [and to pay the Society defense attorney his fee and expenses from funds of this Society within their control, but they shall not pay or obligate this Society to pay any court costs or judgments rendered against any member in the final determination of any case, nor shall they be obligated without affirmative action, to pay the fees of any local attorney employed by the member. The Defense Board shall be empowered to contract with agents or attorneys as it deems necessary, insofar as the amounts to be expended thereby are approved, or do not exceed the amount of the annual budget for defense purposes.] TO ASSIST SUCH MEMBER IN PLANNING FOR DEFENSE AND IF REQUESTED BY THE DEFENDANT AND DEEMED ADVISABLE BY THE DEFENSE BOARD PROCURE THE SERVICES OF AN ATTORNEY APPROVED BY THE KANSAS MEDICAL SOCIETY, PROVIDED THE ENTIRE COST OF SUCH SERVICES SHALL BE PAID BY THE DEFENDANT. THE SOCIETY SHALL NOT BE OBLIGATED TO PAY ANY FEES FOR LEGAL SERVICES, NOR ANY COSTS OR JUDGMENTS ARISING OUT OF ANY ACTION INVOLVING THE PROFESSIONAL SERVICES OF ANY MEMBER.

10. Resolved: That the By-Laws, Chapter IX, Section 6 be amended by deleting the first two sentences and substituting therefore "Section 6. Disbursements for defense to include reimbursement for any actual expenses incident to travel, by members of the Defense Board, required in the performance of official duties, and a per diem sum fixed by the Council for the days actually spent in such official duties may be allowed."

CHAPTER IX—DEFENSE BOARD

Section 6. [Disbursements for defense shall be annually estimated in advance by the Defense Board as based upon average expenses of the preceding two years. The computed amount shall then be included in the annual budget of the Executive Secretary for presentation to and approval by the House of Delegates.] DISBURSEMENTS, FOR DEFENSE TO INCLUDE REIMBURSEMENT FOR ANY ACTUAL EXPENSES INCIDENT TO TRAVEL, BY MEMBERS OF THE DEFENSE BOARD, REQUIRED IN THE PERFORMANCE OF OFFICIAL DUTIES, AND A PER DIEM SUM FIXED BY THE COUNCIL FOR THE DAYS ACTUALLY SPENT IN SUCH OFFICIAL DUTIES MAY BE ALLOWED.

The remainder of the section will remain but has no bearing upon the amendment so is not reprinted here.

COMMENT: These two amendments were prepared at the request of the Council. Reasons for this change from the original plan of providing services of the attorney for the Society when requested can best be explained by them.

The committee recommends adoption of these two amendments.

---

11. Resolved: That the By-Laws, Chapter II, Section 1 be amended by striking out the words "Twenty-five" and substituting the word "Fifty."

CHAPTER II—ASSESSMENTS

Section 1. The amount of the annual assessment for The Kansas Medical Society only shall be not more than [twenty-five] FIFTY dollars per member, the exact amount to be determined by the Council after consideration of the annual budget for the ensuing year and to be announced to the various component societies not less than three months before the beginning of each fiscal year. . . .

The remainder of this section has no bearing on this amendment.

COMMENT: Increased activities and increased costs generally have made expenses exceed the income for the past few years. Rather than delve into reserve funds established years ago, this increase of dues is recommended. While fifty dollars is set as a maximum, it is presumed that a lesser sum may be set by the Council annually.

The committee recommends adoption of this resolution.

---

12. Resolved: That the By-Laws, Chapter I, Section 4 be amended by deleting the last sentence.

CHAPTER I—MEMBERSHIP

Section 4. . . . [Honorary members having qualifications required by the Constitution of the American Medical Association may be recommended for Affiliate Fellowship in the American Medical Association by vote of the House of Delegates.]

The preceding portion of this section has no bearing on this amendment.

COMMENT: Since fellowship classifications within the American Medical Association have been abolished, this sentence has been rendered inoperative.

The committee recommends adoption of this resolution.

Numbers 13 and 14 pertain to the same subject:

13. Resolved: That the By-Laws, Chapter XI, Section 1a, be amended by adding a new section 37 "Committee on General Practice Award."

14. Resolved: That the By-Laws, Chapter XI be amended by adding a section.

CHAPTER XI—COMMITTEES

SECTION 37. THE COMMITTEE ON GENERAL PRACTICE AWARD SHALL CONSIST OF FIVE OR MORE MEMBERS OF WHICH ONE SHALL BE A COUNCILOR AND ONE THE PRESIDENT OF THE KANSAS ACADEMY OF GENERAL PRACTICE OR A MEMBER OF THAT ACADEMY DESIGNATED BY HIM. TWO OR MORE MEMBERS SHALL HAVE SERVED ON THE RETIRING COMMITTEE. THE DUTIES OF THE COMMITTEE SHALL BE TO DETERMINE AN INDIVIDUAL MEMBER AND SUBMIT ADEQUATE MATERIAL AND INFORMATION IN SUPPORT OF ANY KANSAS MEDICAL SOCIETY MEMBER DESIRED TO BE SUBMITTED TO THE AMERICAN MEDICAL ASSOCIATION COMMITTEE AS A CANDIDATE FOR THE ANNUAL GENERAL PRACTITIONER'S AWARD.

COMMENT: This will establish a permanent mechanism whereby this Society may nominate, if it wishes to do so, a candidate for the American Medical Association award to the outstanding general practitioner of the year.

The committee recommends adoption of these resolutions.

---

15. Resolved: That the By-Laws, Chapter VIII, Section 12, be amended by striking the word "Eleven" and substituting "a majority of the."

CHAPTER VIII—THE COUNCIL

Section 12. [Eleven] A MAJORITY OF THE members of the Council shall constitute a quorum.

COMMENT: This appears advisable to care for this question regardless of the number on the Council.

The committee recommends this be passed.

Numbers 16, 17, 18, and 19 refer to one subject:

COMMENT: Revision of the Council districts in an effort to produce a more equitable distribution of members within the several districts has been a project with which your committee has worked during this past year. The following four plans appear feasible.

The committee recommends that the House of Delegates select its preference from these four alternatives:

Number 16 retains 12 council districts.

Number 17 creates 15 council districts.

Number 18 creates 14 council districts.

Number 19 creates 17 council districts.

16. Resolved: That the By-Laws, Chapter VIII, Section 13, enumerating the counties comprising the present Councilor Districts remain as at present except that Leavenworth County be removed from District 2 and placed into District 1.

CHAPTER VIII—THE COUNCIL

Section 13. The councilor districts shall be comprised of the following counties: First District: Marshall, Nemaha, Brown, Doniphan, Pottawatomie, Jackson, Atchison, [and] Jefferson AND LEAVENWORTH Counties. Second District: [Leavenworth,] Wyandotte, Douglas, Johnson, Franklin, Miami, Anderson and Linn Counties.

The remainder of this long section is not quoted because it remains as at present.

COMMENT: This will occasion no change in Councilor elections.

17. Resolved: That the By-Laws, Chapter VIII, Section 13 enumerating the present Councilor Districts shall retain their present boundaries except that Leavenworth County be placed into District 1 and that three new districts shall be formed whereby Wyandotte County shall be removed from District 2 and become District 13, Shawnee County shall be removed from District 4 and become District 14 and Sedgwick County shall be removed from District 6 and become District 15.

COMMENT: This will alter counties as currently listed in this section but because of its length is not reprinted here. Such action will not conflict with other sections of the Constitution and By-Laws and will require the election of five councilors each year for the future.

If this amendment is voted the following, in addition to the regular Councilor elections, will become necessary during this annual session.

a. District 4 (without Shawnee County) would elect a Councilor for a term expiring in 1957.

b. District 6 (without Sedgwick County) would elect a Councilor for a term expiring in 1956.

c. District 13 (Wyandotte County) would elect a Councilor for a term expiring in 1955.

18. Resolved: That the By-Laws, Chapter VIII, Section 13 be deleted after the word "counties" and the following be inserted:

District 1—Atchison, Brown, Doniphan, Jackson, Jefferson, Leavenworth and Nemaha.

District 2—Anderson, Douglas, Franklin, Johnson, Linn, Miami and Wyandotte.

District 3—Allen, Bourbon, Cherokee, Crawford, Labette, Montgomery, Neosho, Wilson and Woodson.

District 4—Clay, Geary, Marshall, Pottawatomie, Riley and Washington.

District 5—Chase, Coffey, Lyon, Morris, Osage, Shawnee and Wabaunsee.

District 6—Butler, Chautauqua, Cowley, Elk and Greenwood.

District 7—Cloud, Dickinson, Ellsworth, Jewell, Lincoln, Mitchell, Ottawa, Republic and Saline.

District 8—Harvey, McPherson, Marion, Reno and Rice.

District 9—Barber, Harper, Kingman, Sedgwick, and Sumner.

District 10—Ellis, Graham, Osborne, Phillips, Rooks, Russell, Smith and Trego.

District 11—Barton, Hodgeman, Ness, Pawnee, Rush and Stafford.

District 12—Clark, Comanche, Edwards, Ford, Gray, Kiowa, Meade, Pratt and Seward.

District 13—Cheyenne, Decatur, Gove, Logan, Norton, Rawlins, Sheridan, Sherman, Thomas and Wallace.

District 14—Finney, Grant, Greeley, Hamilton, Haskell, Kearny, Lane, Morton, Scott, Stanton, Stevens and Wichita.

COMMENT: If this resolution is adopted, it is recommended that each present councilor retain his position until the expiration of his present term and that new councilors be elected in the newly formed districts, their terms to be staggered as indicated to insure proper rotation.

One conflict is created because two present councilors reside in the proposed District 12. It is recommended that both continue in their office for one year, at which time Doctor Klein will be ineligible for re-election because of having completed two successive terms. Then in 1956, when Doctor Black's second term expires, District 12 will elect a new councilor for a three-year term.

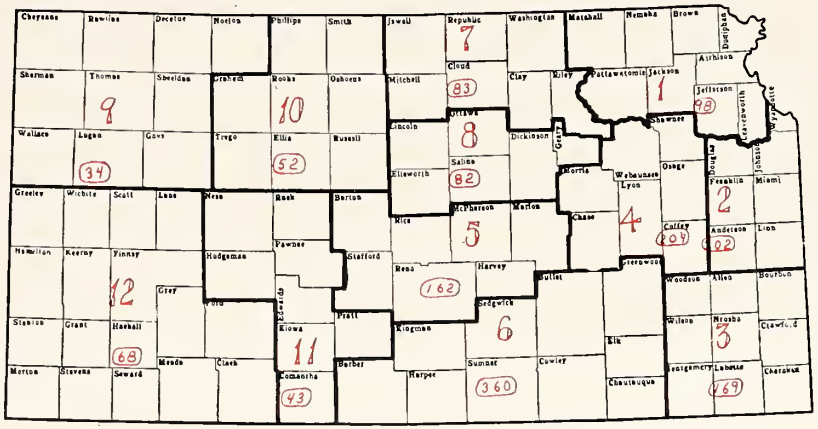
District	*Councilor*	*Term Expires*
1	W. L. Anderson, M.D., Atchison	1954
2	H. P. Jones, M.D., Lawrence	1954
3	Charles E. Vestle, M.D., Humboldt	1955
4	S. A. Anderson, M.D., Clay Center	1954
5	F. C. Taggart, M.D., Topeka	1956
6	NEW	1957
7	A. E. Rueb, M.D., Salina	1954
8	H. M. Glover, M.D., Newton	1956
9	N. L. Francis, M.D., Wichita	1955
10	L. W. Reynolds, M.D., Hays	1955
11	NEW	1955
12	C. V. Black, M.D., Pratt	1956
	R. G. Klein, M.D., Dodge City	1955
13	J. L. Jenson, M.D., Colby	1956
14	NEW	1956

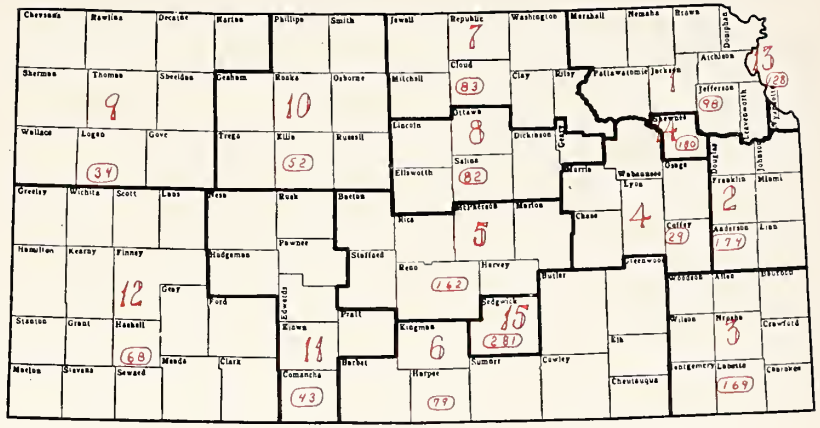
19. Resolved: That the By-Laws, Chapter VIII, Section 13 be deleted after the word "counties" and the following be inserted:

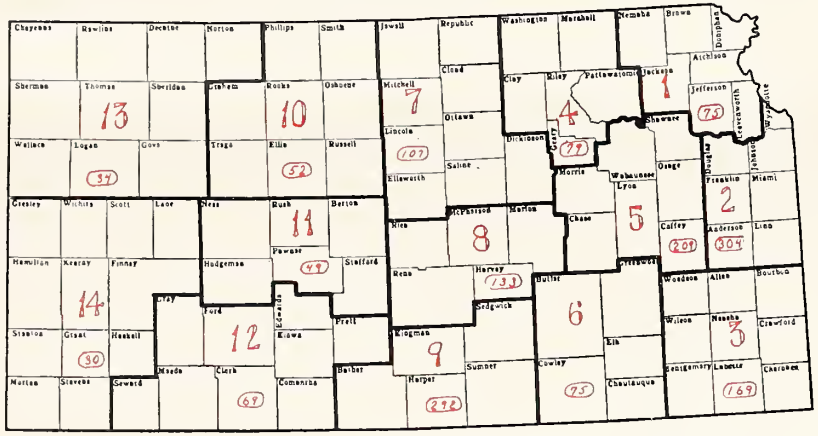
District 1—Atchison, Brown, Doniphan, Jackson, Jefferson, Leavenworth and Nemaha.

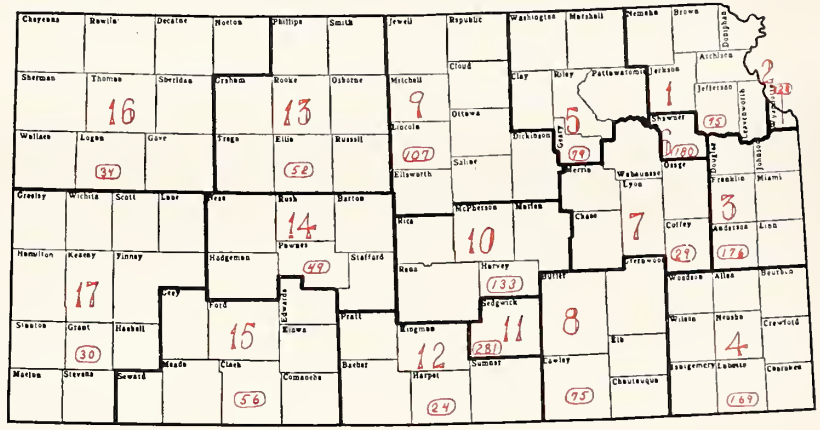
District 2—Wyandotte County.

District 3—Anderson, Douglas, Franklin, Johnson, Linn and Miami.









Map 1, upper left, shows the present districts except for Leavenworth County—see Resolution No. 16.

Map 2, upper right, shows the present districts except for the creation of three additional districts—see Resolution No. 17.

Map 3, lower left, shows 14 districts—see Resolution No. 18.

Map 4, lower right, is similar to Map 3 except for the addition of three districts—see Resolution No. 19.

District 4—Allen, Bourbon, Cherokee, Crawford, Labette, Montgomery, Neosho, Wilson and Woodson.

District 5—Clay, Geary, Marshall, Pottawatomie, Riley and Washington.

District 6—Shawnee County.

District 7—Chase, Coffey, Lyon, Morris, Osage and Wabaunsee.

District 8—Butler, Chautauqua, Cowley, Elk and Greenwood.

District 9—Cloud, Dickinson, Ellsworth, Jewell, Lincoln, Mitchell, Ottawa, Republic and Saline.

District 10—Harvey, McPherson, Marion, Reno and Rice.

District 11—Sedgwick County.

District 12—Barber, Harper, Kingman, Pratt and Sumner.

District 13—Ellis, Graham, Osborne, Philiips, Rooks, Russell, Smith and Trego.

District 14—Barton, Hodgeman, Ness, Pawnee, Rush and Stafford.

District 15—Clark, Comanche, Edwards, Ford, Gray, Kiowa, Meade and Seward.

District 16—Cheyenne, Decatur, Gove, Logan, Norton, Rawlins, Sheridan, Sherman, Thomas and Wallace.

District 17—Finney, Grant, Greeley, Hamilton, Haskell, Kearny, Lane, Morton, Scott, Stanton, Stevens and Wichita.

COMMENT: This varies from resolution No. 10 in the separation of the three large counties and in changing Pratt County.

If this resolution is adopted, the following situation would apply regarding Councilor elections at this session. No conflicts are created.

District	*Councilor*	*Term Expires*
1	W. L. Anderson, M.D., Atchison	1954
2	NEW	1955
3	H. P. Jones, M.D., Lawrence	1954
4	C. E. Vestle, M.D., Humboldt	1955
5	S. A. Anderson, M.D., Clay Center	1954
6	F. C. Taggart, M.D., Topeka	1956
7	NEW	1956
8	NEW	1957
9	A. E. Rueb, M.D., Salina	1954
10	H. M. Glover, M.D., Newton	1956
11	N. L. Francis, M.D., Wichita	1955
12	C. V. Black, M.D., Pratt	1956
13	L. W. Reynolds, M.D., Hays	1955
14	NEW	1955
15	R. G. Klein, M.D., Dodge City	1955
16	J. L. Jenson, M.D., Colby	1956
17	NEW	1957

Respectfully submitted,
A. W. Fegtly, M.D., *Chairman*

Control of Cancer

R. E. Speirs, Dodge City, Chr.; J. P. Berger, Wichita; T. P. Butcher, Emporia; A. M. Cherner, Hays; A. A. Fink, Topeka; H. L. Hiebert, Topeka; W. J. Kiser, Wichita; O. F. Prochazka, Liberal; R. H. Riedel, Topeka; N. P. Sherwood, Lawrence; C. D. Snyder, Winfield; R. E. Stowell, Kansas City; J. F. Thurlow, Hays; G. M. Tice, Kansas City; L. E. Vin Zant, Wichita; K. E. Voldeng, Wellington; H. M. Wiley, Garden City.

To the House of Delegates:

This committee has had three meetings during the year, two at Wichita and one at Topeka. Membership response was excellent, and all members of the committee took an active part in carrying out their various duties.

The most outstanding activity of the committee was the planning of the Mid-West Cancer Conference held in Wichita on April 1 and 2 at the Broadview Hotel. The great part of this work was carried on by just a few members of the committee, and certainly they should be extended a vote of appreciation for the effort and time spent in bringing together an excellent array of speakers. Particular appreciation is due Dr. H. M. Wiley, who served as chairman of the program committee and spent many hours arranging the event, and Dr. J. P. Berger, who directed local arrangements in Wichita and arranged all publicity.

The Committee on Control of Cancer also serves as the Medical and Scientific Committee for the American Cancer Society, Kansas Division. As a result of this connection, much time was spent at working out various problems which were referred from the state cancer society office. The action taken is summarized below.

The committee approved continuation of annual payments to hospitals that have diagnostic cancer clinics. Mr. Miles Pulford, executive director of the Kansas Division, was asked to summarize reports from these clinics.

Also approved was a motion that the program of professional education carried on by the Kansas Division through literature mailed to all physicians in the state be continued. Other programs which were heartily approved for continuation are the Mid-West Cancer Conference, financial support to the University of Kansas School of Medicine for postgraduate courses in specialties having to do with cancer, financial assistance to the Kansas State Board of Health and the Kansas Tuberculosis and Health Association for the mass chest x-ray survey program in Kansas, provision for scholarships for nurses, occupational therapists, laboratory technicians, etc., support of the Kansas State Board of Health effort to maintain a cancer registry, and support of the University of Kansas School of Medicine Tumor Registry and follow-up service.

The committee studied proposals to assist institutions in obtaining deep therapy machines and radium and to support isotope programs throughout the state. Both proposals were rejected.

During the past year the committee attempted to draw up a policy for the guidance of the Kansas Division. This was a difficult undertaking because the requests submitted to the Kansas Division vary from month to month. Some progress was made in outlining this guidance, but the door was left open for changes required by advancements in the field of medicine.

The following seven projects are recommended for the consideration of the Committee on Control of Cancer during the coming year: improving the content of the *Cancer News,* expansion of local unit organization, development of film on cancer in Kansas, promotion of a television program for lay education, provision for giving aid to cancer patients, development of television for medical student education, and study of the cancer problem in rural areas.

The chairman wishes to take this opportunity to thank all members for their splendid co-operation during the past year.

Respectfully submitted,
R. E. Speirs, M.D., *Chairman*

Control of Tuberculosis

A. L. Ashmore, Wichita, Chr.; Andre Baude, Topeka; F. E. Bishop, Atwood; H. L. Bogan, Baxter Springs; R. I. Canuteson, Lawrence; W. G. Rinehart, Pittsburg; C. F. Taylor, Norton; F. A. Trump, Ottawa; C. J. W. Wilen, Manhattan.

To the House of Delegates:

This committee has not held a meeting during the past year, so has no report to submit.

Respectfully submitted,
A. L. Ashmore, M.D., *Chairman*

Emergency Medical Care

D. P. Trees, Wichita, Chr.; W. H. Algie, Kansas City; W. A. Grosjean, Winfield; G. W. Hammel, El Dorado; P. B. Leffler, Pittsburg; J. W. Manley, Kansas City; J. M. Mott, Topeka; W. A. Smiley, Jr., Junction City; Nathaniel Uhr, Topeka.

To the House of Delegates:

The Committee on Emergency Medical Care has no report to make at this time.

Respectfully submitted,
D. P. Trees, M.D., *Chairman*

Endowment

J. W. Randell, Marysville, Chr.; G. H. Jackman, Cimarron; F. D. Murphy, Lawrence; E. H. Terrill, Wichita.

To the House of Delegates:

The principal project of this committee has been an effort to obtain contributions from the membership for the University of Kansas School of Medicine. A goal of $50,000 has been set to be raised during the summer months for this project. Your committee respectfully requests the endorsement of this project by the House of Delegates and the individual support of each member toward the accomplishment of this necessary and worthwhile endeavor.

A full explanation would require considerably more space than can be allotted at this time. Therefore, the following represents a condensed outline of only the most salient facts.

Medical education must be subsidized. If this is not accomplished through voluntary contributions, it will be undertaken by government, and the resultant interference and control and obligation will immediately be obvious to everyone.

The physician paid only a fraction of the cost of his medical education and therefore has an obligation not only to the school from which he was graduated but to the school in this state where he earns his livelihood. The fact that such contributions are deductible from income tax declarations should increase the amount to be given.

Your committee, through co-operation with Dr. C. V. Black of Pratt, state chairman of the American Medical Education Foundation, has evolved a plan whereby contributions may be made to cover both projects at one time. A check made out to the American Medical Education Foundation and sent to the American Medical Association in Chicago may be earmarked for use by the School of Medicine at the University of Kansas, and, through that procedure, credit will be obtained in the foundation and the entire contribution will be forwarded to the University of Kansas. Checks may be made in any denomination, but it can quickly be recognized that they should be substantial.

Some states collect these contributions on the basis of an assessment required of each member. Your committee begs the consideration of this body for the suggestion that a special assessment be added to the dues of the Kansas Medical Society for the purpose of making a sizable contribution to the University of Kansas School of Medicine. It is felt that this assessment should not be too large but that individual gifts should be made in addition.

The A.M.E.F. recently made its annual report and during 1953 raised more than $1,000,000. A total of 17,564 physicians contributed $467,217.50. Among the states nearby, some contributed less than did Kansas, but a large majority gave far more. For example,

523 Colorado doctors contributed to this fund; 163 gave out of Nebraska; Illinois led the nation with 8,472 doctors contributing. In Kansas, 54 doctors gave to the A.M.E.F. during 1953, but the total sum contributed through this source was only a little over $2,000. And this represents gifts to all schools!

Actually, Kansas is credited with $12,641, of which $10,000 represents a gift from the national auxiliary which came under the name of Mrs. Schaefer, the president. Seventeen county and city auxiliaries contributed approximately $500. To further show the Kansas situation in this regard, the following figures will indicate that our state has consistently fallen well below the national average. In 1952, 3.48 per cent of all doctors contributed to A.M.E.F., but in Kansas the average stood at .85 per cent. In 1953, the national average rose to 8.89 per cent and in Kansas went to 2.42 per cent.

A number of physicians contributed directly to the University of Kansas whose gifts were not credited by the A.M.E.F. You will recall previous communications concerning the Continuation Study Building now being completed on the campus of the University of Kansas School of Medicine. This building will house a magnificent auditorium which will seat approximately 900 persons. There will also be a library, cafeteria, and attractive living quarters. This building has been constructed through funds donated to the school. The Kress Foundation gave $150,000. From the Battenfeld Foundation a total of $104,845 was given. Business and industry in Kansas City contributed another $135,750.95. The University of Kansas Endowment Association gave $80,000.

During the past years, while this building was being planned, contributions in varying amounts have been received from numerous other sources, as for example proceeds from the book store, almost $18,000; student fees, $10,000, and contributions from others than doctors, $16,500. During this time, doctors from outside Kansas have given $30,537, and Kansas doctors have given $44,644.96.

While the above certainly shows an interest, it is far below not only what is needed to adequately furnish this building but represents a sum far below that which Kansas physicians might be expected to contribute. Therefore, your committee is now undertaking a special project to add an additional $50,000 for this purpose and sees the opportunity for combining one gift in the service of both the A.M.E.F. and the Continuation Study Building at the University of Kansas School of Medicine.

Nor is this utterly unselfish, because this building will be used for postgraduate work, and every doctor in Kansas will be individually benefited through its functions. We believe in this project and recognize that a large segment of the Society must become actively engaged in the effort to insure its success. Therefore, may we respectfully submit the following proposals for approval and the active endorsement of the House of Delegates.

(1) That the Kansas Medical Society raise $50,000 between this date and September 1, 1954; (2) that the House of Delegates consider the possibility of making a small contribution required in the form of a special assessment from each member; (3) that additional larger individual contributions be actively solicited through appointed representatives in each county medical society; (4) that the contributions be given to the A.M.E.F. but designated as gifts to the University of Kansas School of Medicine.

The adoption of the above program will produce tangible evidence of the interest physicians in Kansas have in maintaining the freedom of medical education. Such contributions will, in addition to that, complete the Continuation Study Building at the University of Kansas in which each contributor may not only take pride but from which he will derive a very large personal benefit.

Respectfully submitted,
J. W. Randell, M.D., *Chairman*

Expert Testimony

C. E. Joss, Topeka, Chr.; E. J. Frost, Wichita; J. L. Lattimore, Topeka; C. D. McKeown, Wichita.

To the House of Delegates:

Again, as in past years, this committee has stood ready to assist the jurors and barristers of Kansas in evaluating the accuracy of medical testimony delivered in the courts. Again, as during past years, the committee has not been called upon for assistance and therefore continues to stand by and has no report to make.

Respectfully submitted,
C. E. Joss, M.D., *Chairman*

History

R. R. Melton, Marion, Chr.; A. C. Gulick, Goodland; H. E. Haskins, Kingman; C. S. Huffman, Columbus; Alfred O'Donnell, Ellsworth; O. D. Walker, Salina.

To the House of Delegates:

The danger of the irrevocable loss of historical material through the passage of time increases with each year. The approaching centennial of the Kansas Medical Society represents an occasion during which a history will be most useful. Therefore, this committee believes the House of Delegates should act during this session to prepare a systematic plan whereby historical data may be preserved and a history written.

Your committee, after deliberation upon this problem, recognizes that this project cannot be undertaken effectively if entirely conducted as a leisure time activity of the doctors of medicine in active practice. It cannot be a successful venture without the expenditure of money. Recognizing that a sizable appropriation will not be practical at this time, your committee recommends the following as at least a partial solution to this question which, if not taken now, will shortly become impossible regardless of how much money may be available in the future.

The first point is that the Society invest in a steel file into which may be collected all articles and items of historical interest to the Society. This is for the purpose of providing a central storage space for such material only.

Point 2 is that the membership be advised about this project and all be invited to contribute whatever information or material they have at their disposal that might be pertinent to the subject.

The third suggestion is that the executive secretary be empowered to employ a person who will gather historical material, at least on a part-time basis. As a tentative suggestion, an employee of the Kansas Historical Society might be willing to work a few hours each week upon this project and should be paid by the Kansas Medical Society for such services.

Point 4 is that a more formal and perhaps a more remunerative effort might be employed through the offer of a scholarship to a graduate student who will undertake the history of the Kansas Medical Society as a thesis. It is possible that this might be undertaken in several sections by different students, as for example, different periods of Kansas history. Or, it might be undertaken topically, as for example, the development of the Kansas State Board of Health, the Kansas State Board of Medical Registration and Examination, etc. It is known that Emporia State Teachers' College conducts an authorized course in library science. However, the thesis might be equally appropriate out of other schools in such subjects as sociology, history, journalism, and others. The amount of such scholarships should be of sufficient size to create an interest, but since the student presumably would thereby be receiving credit toward an academic degree, the value received by the Kansas Medical Society would greatly exceed its cost.

Respectfully submitted,

R. R. Melton, M.D., *Chairman*

Hospital Survey

A. C. Armitage, Hutchinson, Chr.; P. L. Beiderwell, Belleville; M. E. Christmann, Pratt; A. P. Cloyes, El Dorado; E. R. Gelvin, Concordia; L. C. Hays, Cedarvale; J. L. Jenson, Colby; A. L. Nichols, Hiawatha; P. A. Petitt, Paola; A. J. Rettenmaier, Kansas City; Joe Seitz, Jr., Wakeeney; F. C. Shepard, Clay Center.

To the House of Delegates:

At a meeting of this committee a review was given of the rules governing Kansas convalescent infirmaries. Representatives of the Kansas State Board of Social Welfare attended the meeting to clarify some of the regulations set forth.

Mr. Roy Johnston, from the Kansas State Board of Health, also attended the meeting and reported that they are making a hospital survey at present. The committee believes that the trend of hospital construction should be directed toward the larger cities. They find several rural areas now presented with the problem of an oversupply of hospital beds. Kiowa, Kansas, has been suggested as a community which has an ideal hospital for the size of the community. The committee looks forward to extending this project into the coming year.

Respectfully submitted,

A. C. Armitage, M.D., *Chairman*

Industrial Medicine

M. A. Walker, Kansas City, Chr.; J. W. Cavanaugh, Topeka; J. A. Grove, Newton; C. W. Hall, Hutchinson; H. R. Hodson, Wichita; E. S. Miller, Kansas City; H. L. Regier, Kansas City; R. W. Urie, Parsons.

To the House of Delegates:

Several items have been brought to the attention of this committee and have been taken care of by telephone or written communication. A meeting of the committee will be held in Topeka on March 21, after the date of the deadline for committee reports.

One of the problems was the failure of several physicians in the state to understand the formalities and legal aspects of the examination of patients in connection with Workmen's Compensation matters. These physicians had declined to allow the attorney for a plaintiff to be present during the interrogation of the patient, on the basis that no third party, particularly a layman, had a right to be present during an examination.

This matter was discussed by phone with Mr. Simeon Webb, deputy commissioner. The legal aspect of this situation is that the examination of a patient for legal purposes consists of two separate portions. The first is the interrogation, in the physician's consulting room, which may properly be conducted in the presence of the plaintiff's attorney. The patient and the physician then go to the examining room, without the attorney, for the physical examination.

The patient may, however, have his own physician present at this examination if he so desires.

A questionnaire on medical relations under Workmen's Compensation was received from the Council on Industrial Health of the American Medical Association. One question was: What difficulties have members of the medical profession in your state experienced in the treatment and examination of injured employees under the Federal Employees' Compensation Act? The consensus of the members of our committee was that medical work done by us under this act is quite unsatisfactory in many respects, particularly the slow pay due to government red tape, antiquated lists of designated physicians, inflexibility and inadequacy of facilities for consultation, and complicated forms.

The questionnaire also inquired as to whether the same difficulties were encountered under our state Workmen's Compensation Act governing employees of private corporations. The general belief of our committee was that they had found the Kansas State Workmen's Compensation Act quite satisfactory.

Respectfully submitted,

MAURICE A. WALKER, M.D., *Chairman*

MATERNAL WELFARE

David E. Gray, Topeka, Chr.; D. A. Anderson, Salina; R. M. Carr, Junction City; L. E. Filkin, Concordia; H. M. Floersch, Kansas City; H. M. Foster, Hays; R. G. Heasty, Manhattan; R. L. Hermes, Lawrence; G. M. Martin, Topeka; Robert L. Newman, Kansas City; C. D. Schrader, Newton; Robert Sohlberg, Jr., McPherson.

To the House of Delegates:

During the past year your Committee on Maternal Welfare has had three meetings. At the first meeting representatives from four adoption agencies were present, and each stressed the importance of placing all children eligible for adoption in the hands of an agency qualified to place a child. It was pointed out that about 20 per cent of the children eligible for adoption, other than those adopted by relatives, are being placed by physicians.

The committee felt that physicians would be happy to co-operate with such agencies if they were better informed on procedures. It was suggested that the agencies and the committee unite in conducting an educational program. The committee felt that a report in the JOURNAL, a letter to the membership giving general information on adoptions, and the encouraging of county medical societies to discuss adoption proceedings would result in a better understanding. An article in the JOURNAL in September, "The Physician's Role in Adoptions," drew favorable comments.

Considerable time was spent reviewing maternal deaths in Kansas. All maternal deaths through 1953 were discussed, and the committee has asked for further information on several of these. Various methods for securing more full and accurate information on maternal deaths are being considered.

The committee approved a recommendation that the Kansas State Obstetrical Society appoint doctors from various districts in Kansas to obtain information on maternal deaths when requested, and, in so far as possible, it recommends that members of this committee be appointed for this purpose. It was also recommended that this committee select interesting and unusual cases of maternal deaths for papers to be published in the JOURNAL.

The committee approved a regulation concerning the use of silver nitrate for the treatment of eyes in newborn infants.

Respectfully submitted,

DAVID E. GRAY, M.D., *Chairman*

MEDICAL ASSISTANTS

H. J. Davis, Topeka, Chr.; J. J. Basham, Eureka; O. W. Davidson, Kansas City; A. E. Hiebert, Wichita; Howard Kennedy, Topeka; George C. Meek, Arkansas City.

To the House of Delegates:

This committee met several times during the year to assist the Kansas Medical Assistants' Society in the operation of its organization. The committee has endeavored to advise them concerning program possibilities and has attempted to encourage the organization of local societies in areas that do not now have them.

Dr. W. F. Bernstorf, past president of this Society, submitted a project to this committee which has received the endorsement of the Council and is hereby presented for consideration by the House of Delegates. It appears that there is no readily available school in which medical assistants may learn the techniques of office management, telephone courtesy, and the thousand other responsibilities attendant to their duties. Her success in her employment, therefore, depends upon her ability, experience, and the inspiration she receives within her own office, and through her association with others in similar work. It appears that a short annual academic course in subjects of this type might benefit her service to the physician for whom she works.

It is planned, then, should this meet with the approval of the House of Delegates, to arrange a brief course of study consisting of possibly two days and presenting the same program at the University of Kansas in Lawrence for the eastern half of the state and at Hays State Teachers' College for western Kan-



Through its probable action on the labyrinth, dependable control of vertigo and nausea has made Dramamine the most widely-prescribed product in its field.

Vertigo: The Labyrinthine Structure and Dramamine®

Dramamine's remarkable therapeutic efficiency is believed to be the result of suppression of the over-stimulated labyrinth. Thus it prevents the resulting symptom complex of vertigo, nausea and, finally, vomiting.

First known for its value in motion sickness, Dramamine is widely prescribed for nausea and vomiting of pregnancy, electroshock therapy, certain drugs and narcotization. It relieves vertigo of Ménière's syndrome, fenestration procedures, labyrinthitis, hypertensive disease and that accompanying radiation and antibiotic therapy.

A most impressive number of clinical studies shows that Dramamine has a high therapeutic index and minimal side actions. Drowsiness is possible in some patients but in many instances this side action is not undesirable.

Dramamine (brand of dimenhydrinate) is available in tablets of 50 mg. each; liquid containing 12.5 mg. per 4 cc. Dramamine is accepted by the Council on Pharmacy and Chemistry of the American Medical Association. G. D. Searle & Co., Research in the Service of Medicine.

sas. The faculty would consist of selected members of the Kansas Medical Society and possibly other business or professional executives, faculty members of the schools involved, and some outside speakers. The course would be designed for instruction rather than for entertainment, and some additional individual research upon selected subjects would be recommended. Upon completion of this course, a certificate would be awarded.

The present deterring factor is finance. It was felt that this project, if of value at all, would be of benefit to the individual physician. For that reason, the committee recommends that the physician should send the student to this course and pay her expenses. If conducted on an annual basis, the project might become a means whereby medical assistants might be instructed to render a better service in all areas of this state than has been possible at the present while no formal instructive courses are available.

Respectfully submitted,

H. J. Davis, M.D., *Chairman*

Medical Economics

B. A. Nelson, Manhattan, Chr.; J. N. Blank, Hutchinson; R. L. Gench, Fort Scott; E. J. Grosdidier, Kansas City; D. H. Macrae, Topeka; G. E. Milbank, Wichita; M. B. Miller, Topeka; F. A. Moorhead, Neodosha; L. S. Nelson, Jr., Salina; R. Solhberg, Jr., McPherson.

To the House of Delegates:

Your Committee on Medical Economics has for some years worked in two separate although related fields. Both deal with economics, both represent projects designed to be of assistance to the membership, but one is strictly personal while the other is professional. On the latter, of course, there is a combined interest in the physician and his patient alike.

In previous years your committee arranged to make available health and accident insurance at a cost substantially below the prevailing rate required of individual contracts and in addition succeeded in obtaining a waiver of proof of insurability. Therefore, this personal benefit resulted not only in a considerable financial savings but made such protection available to a sizable number of members who, except for this opportunity, could not purchase health and accident insurance at any price.

The first of these projects came with the Commercial Insurance Company serviced in northeast Kansas by Mr. W. H. Schlatter, 1324 South 32nd Street, Kansas City, Kansas, and in the remainder of the state by Mr. C. Ray Tyler, Brown Building, Wichita. At first this was a small basic policy which has, in its 10 years of experience with Kansas physicians, been repeatedly expanded until today the benefits are considerably broader than in the beginning.

General acceptance of this Society benefit and the often expressed desire for still larger limits of coverage prompted your committee to again explore the field and two years ago recommended the Washington National Insurance Company, of which Mr. James C. First of Topeka is the state representative. Now the physician has available two policies to choose from or, as is recommended by the committee, he may carry both, thereby assuring himself a higher degree of protection against loss of income because of illness than could be obtained in any single policy. Both are sold at less than the cost of individual purchase, and both companies accepted, at the time of original enrollment, all applications regardless of the physical condition of the member.

Your committee met with representatives of both the above companies during this year and recommended further broadening of benefits. It is our belief that this will soon be accomplished, that the Commercial Insurance Company may make available a family policy in addition to the present contract, and that the Washington National Company may increase its weekly benefits and lengthen the period for which benefits are available. Further details will be given in direct announcements from these companies.

Since increased benefits are the result of profitable experience which comes only through a broad spread of risk, your committee believes that the Society should exert some effort in recommending these programs to the membership. Both companies offer attractive policies and, if a higher percentage of the total membership takes advantage of this opportunity, the committee believes they will still increase their benefits beyond today's contracts.

Therefore, this committee recommends that the House of Delegates explore whether there are now sufficient members who wish to purchase this protection to justify a request for a period of reopened enrollment. It should be noted that a sizable demand must be evidenced before such a procedure would be practical.

The second project likewise represents an individual economic benefit which, although still in the planning stage, now appears to be possible, pending only whether the membership is sufficiently interested. This pertains to group life insurance. Your committee now has proposals from several companies and will obtain others that enable members of the Kansas Medical Society to purchase life insurance without proof of insurability and at a price considerably under the cost of comparable individually purchased insurance. Success will depend upon a relatively high per cent of participation.

Details can be given, but since a variety of formulas are available which can be selected at a later date,

50 *and seven*

YEARS TREATING ALCOHOL AND DRUG ADDICTION

In 1897 Doctor B. B. Ralph developed methods of treating alcohol and narcotic addiction that, by the standards of the time, were conspicuous for success.

Twenty-five years ago experience had bettered the methods. Today with the advantages of collateral medicine, treatment is markedly further improved.

The Ralph Sanitarium provides personalized care in a quiet, homelike atmosphere. Dietetics, hydrotherapy and massage speed physical and emotional re-education. Cooperation with referring physicians. Write or phone.

The

RALPH

SANITARIUM

A Unit of the Benjamin Burroughs Ralph Foundation for Medical Research

Telephone Victor 3624



529 HIGHLAND AVENUE • KANSAS CITY 6, MISSOURI

Ralph Emerson Duncan, M.D., Medical Director.

these will not be recorded here. This committee believes that the immediate problem is to determine whether enough of the physicians in this Society will purchase low cost life insurance to make further effort in this direction practical. Therefore, the committee will discuss this in more detail at the meeting but begs the House of Delegates to act in directing future activities in this regard.

The third project occupying the attention of the Committee on Medical Economics pertains to malpractice insurance. Current problems in this field represent a complex and bewildering picture of nationwide import. Many specialized interests converge to prohibit any easy solution. Involved are not only the physician and his patient but attorneys, insurance companies, sociological and economic factors and, in fact, other physicians, families or friends of the patient, and the law itself.

Although the solution is not easy, your committee believes a practical method of improving the present situation may be found. This includes for the physician such intangibles as the necessity for the utmost care in professional practice and the strictest voluntary adherence by all physicians to the Code of Ethics. It implies the necessity for generally improved professional-patient relationships and presumes that a better understanding can be achieved among other professional fields. It actually requires much more than this which, if permitted, your chairman begs leave to report in a supplementary discussion wherein certain specific suggestions can be made.

Respectfully submitted,

B. A. Nelson, M.D., *Chairman*

Medical Schools

G. G. Whitley, Douglass, Chr.; J. O. Austin, Garden City; Ralph Ball, Manhattan; R. M. Carr, Junction City; W. J. Feehan, Kansas City; A. C. Hatcher, Wellington; V. H. Hildyard, Baldwin; R. H. Maxwell, Wichita; R. C. Polson, Great Bend; R. E. White, Garnett.

To the House of Delegates:

Since no new problems came to the attention of this committee during the past year, the extensive report made to the House of Delegates in 1953 will not be repeated. This is rather a supplement designed to complete what had been started a year ago.

It is the opinion of this committee that relations between the Kansas Medical Society and the University of Kansas School of Medicine are currently at the best level of any time in history. This is not intended to imply that conditions had ever been critical, because such a statement would not be true. The committee is of the opinion, however, that a considerable improvement has been made recently and can cite numerous examples to support this opinion. The fact that your committee has received no specific complaints is such an example, as is a similar impression voiced by the faculty of the school.

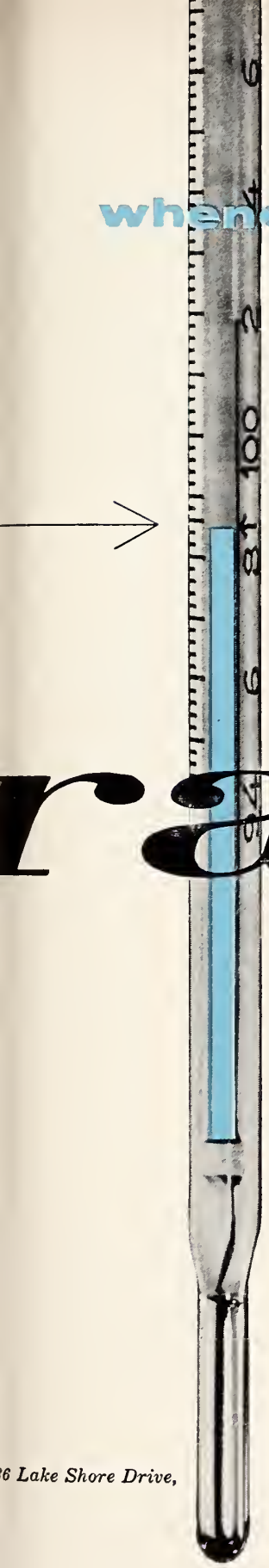
The continually increasing attendance of Kansas physicians at the graduate programs at the university also illustrates this fact and suggests that there may have occurred during the past year a better understanding than ever before. Therefore, the committee is pleased to report that no problem of serious consequences appeared to confront this committee and wishes to suggest that the House of Delegates express its approval of the current operating policies at the medical school.

The committee held a joint meeting with the Committee on Rural Health at the University of Kansas School of Medicine, at which time the dean and a number of department heads were present. Numerous topics were discussed, among which the following are possibly the most significant.

During the past year Dr. Vernon Wilson was employed as Assistant Dean in Charge of Student Affairs. Among his many duties will be maintaining a direct contact with students, which service will enable the profession to correlate such things as locations and many other factors more closely than in the past. It is recommended that the profession become acquainted with Dr. Wilson, whose presence can represent a worthwhile addition to the co-operative program between the Society and the school.

A magnificent Continuation Study Building is nearly completed on the campus. This will provide a distinctive new service to the practicing physician in this state in that meeting facilities as well as housing are included which will make this building of particular use to members of the profession attending graduate courses. Details concerning this building and a project in relation to equipping the Continuation Study Building are reported by the Committee on Endowment, which program is endorsed by this committee.

Dean Wescoe previously reported through the Journal (issue for February 1953 and issue for March 1954) concerning the future building program for the University of Kansas School of Medicine. In spite of the considerable expansion, there still remain areas that need to be improved. The Committee on Medical Schools recommends to the Society that some means be found whereby active assistance may be given the project of enlarging needed facilities. It is felt that an additional three million dollars can construct all necessary facilities to complete the school without further construction during the foreseeable future. To be successful, such a program must receive legislative support, and since it is actually the culmination of a project started some five years ago it is felt that the Society should continue its support toward this end.



whenever you take a temperature think of Tetracyn . . .

". . . in patients with pneumococcal pneumonia, surgical infections, or urinary tract infections . . . oral administration . . . is followed by rapid clinical response. *Symptoms, including fever, largely cleared up within 24 to 48 hours.*"*

racyn

HYDROCHLORIDE

brand of **tetracycline** hydrochloride

**English, A. R., et al.: Antibiotics Annual (1953-1954), New York, Medical Encyclopedia, Inc., 1953, p. 70.*

Supplied: Tetracyn Tablets *(sugar coated)*
250 mg., 100 mg., 50 mg.

6 Lake Shore Drive, Chicago 11, Illinois

Dean Wescoe reported to the committee at some length upon a temporary but a very real problem facing the medical school. He stated at the outset that although serious in Kansas and of great concern, this problem is more acute in many other locations. It came somewhat as a surprise to learn that for the first time the medical school lacks qualified applicants for the student body. This year the freshman class could not be filled. It appears that next year the situation will be even worse. There were 300 applicants, but only approximately 100 had qualifications necessary for admittance. The dean suggested that this may be an aftermath of the depression and the years during which the national birth rate was low. In five years or so, this situation will be reversed, but for the present the medical profession should look for qualified students and encourage them to enter the field of medicine. The dean recommended lower tuition for out-of-state students and suggested a change in the formula whereby more out-of-state students would be selected.

Financial problems of students might be a contributing factor. The committee recommends to the House of Delegates that some committee be appointed to explore the establishment of a loan fund which might be used to aid students who could not otherwise enter the field of medicine. The committee is aware of previous gifts by the medical society for such purposes, but urges this to be made a continuing project which might eventually create a sizable revolving fund.

The Committee on Rural Health was particularly interested in the preceptorship program, which discussion will be reported by that committee. It should be mentioned here also, however, that as a result of this meeting the Dean's Advisory Committee will henceforth include a member from the Committee on Rural Health. There will be reactivated the Dean's Hour at the medical school each Thursday. On this occasion, medical students will hear lectures on topics related to the practice of medicine as distinguished from its scientific aspects once each week. Such topics as ethics, insurance, malpractice, etc., will be presented either by members of the faculty, by practicing physicians in the state, or by specialists in specific fields relating to medicine.

A third innovation is what is known as the Dames' School. This consists of regularly scheduled meetings for the wives of medical students. During such meetings plans were made toward having wives of practicing physicians speak concerning the family life of the doctor, their recreation, their social, religious, and educational experiences. The advantage of this is the experience that many physicians who move from their selected locations do so upon the insistence of their wives.

Two principal topics were discussed, both of which were inconclusively presented in the report of last year. The first of these is the problem of referrals. The dean stated that this represents a most difficult problem. The policy is as strict as it can be made. Patients must be referred by another physician, except in the field of obstetrics because normal deliveries are required for teaching purposes. However, the University of Kansas Hospitals are tax supported institutions and are legally available to taxpayers. Citizens who insist upon being treated at the medical center must be accepted. Therefore, the problem turns about that point and can be successfully controlled only to the degree in which an understanding of the problem exists between the profession practicing within the state and the faculty at the school.

That, in fact, represents the second major remaining difficulty. In spite of improvement which was noticeable in many directions, there still is a pronounced lack of understanding on the part of the profession regarding policies at the school. It is felt that in spite of progress in this direction, continued effort is necessary to acquaint each doctor regarding all activities at the school. Some suggested a monthly news letter, others that announcements concerning policies and activities of the school be posted in the hospital staff rooms regularly. It was finally determined by the committee that a continuing stream of information about the medical school should somehow reach the profession. The committee requests that the House of Delegates authorize the Council to accept responsibility for inaugurating such a program according to whatever means may best accomplish the desired result.

The success of this meeting prompted the decision that once each year the Committees on Medical Schools and on Rural Health meet jointly with the dean and department heads at the University of Kansas School of Medicine.

Respectfully submitted,

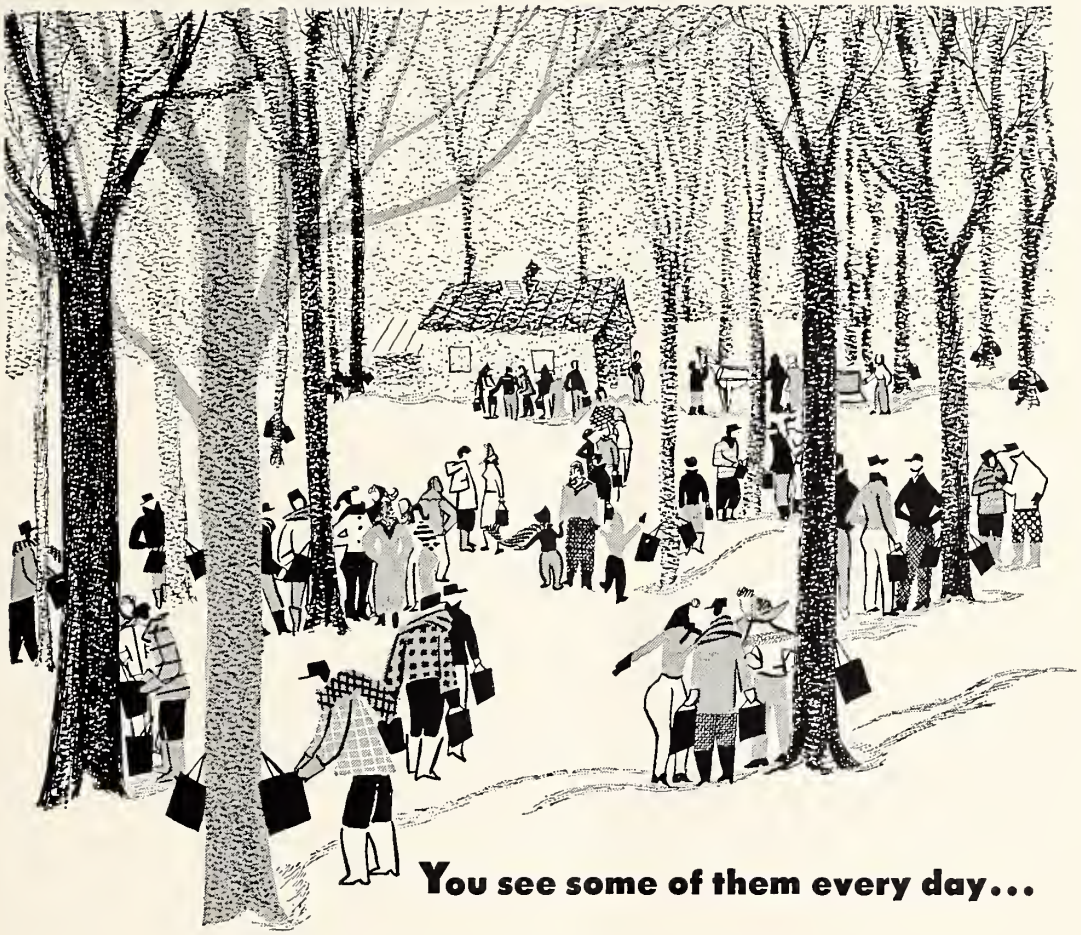
G. G. WHITLEY, M.D., *Chairman*

MENTAL HEALTH

D. B. Foster, Topeka, Chr.; A. J. Adams, Wichita; H. V. Bair, Parsons; R. L. Drake, Wichita; T. L. Foster, Halstead; Mary Glassen, Phillipsburg; E. D. Greenwood, Topeka; L. W. Hatton, Salina; C. C. Hawke, Winfield; George Jackson, Topeka; W. F. Roth, Jr., Kansas City; D. R. Wall, Wichita; M. E. Wright, Lawrence.

To the House of Delegates:

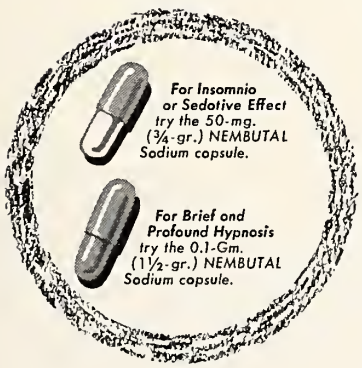
Resolutions passed by the 1953 session of the House of Delegates instructed the Committee on Mental Health to press for improvements in the medical and legal management of sufferers from epilepsy



You see some of them every day...

all the patients who represent

the 44 uses for short-acting NEMBUTAL®



404080A

● As a sedative or hypnotic *in more than 44 clinical conditions,* short-acting NEMBUTAL has established a 24-year-old record for acceptance and effectiveness. Here's why:

1. *Short-acting* NEMBUTAL (*Pentobarbital, Abbott*) *can produce any desired degree of cerebral depression—from mild sedation to deep hypnosis.*
2. *The dosage required is small—only about one-half that of many other barbiturates.*
3. *There's less drug to be inactivated, shorter duration of effect, wide margin of safety and little tendency toward morning-after hangover.*
4. *In equal oral doses, no other barbiturate combines quicker, briefer, more profound effect.*

Any wonder, then, that the use of short-acting NEMBUTAL continues to grow each year. How many of short-acting NEMBUTAL's 44 uses have *you* tried? Abbott

and to continue to press for improvements in the care of the institutionalized mentally ill.

Over a period of eight years, the Committee on Mental Health has participated in a number of important changes in the Kansas statutes leading to improvement in the care of the mentally ill. This continuing program of pressure to further improve legislation has been carried forward this year, in collaboration with the Kansas State Board of Social Welfare's Commission on Institutional Management and the Mental Hygiene Section of the Kansas State Board of Health. This program includes recommendations for changes in the laws which relate to epilepsy.

Exploratory efforts are being made for a program of lay education on mental health.

Other projects of the committee have included recommendations for improving the inspection of the care of the senile and mentally ill in nursing homes, emphasis on the need to plan for additional mental hospital beds in the state of Kansas, efforts to institute a training program for child psychiatrists, advice on staffing patterns in state institutions, a discussion of recommendations on Blue Shield fee schedules as they relate to mental illness, measures to encourage the development of research in mental illness, and co-operative measures to assist the Kansas State Commission on Alcoholism to carry out its functions.

Respectfully submitted,

D. Bernard Foster, M.D., *Chairman*

Necrology

J. F. Gsell, Wichita, Chr.; L. J. Brethour, Junction City; F. D. Lose, Madison; Alfred O'Donnell, Ellsworth; M. O. Steffen, Great Bend; A. E. Titus, Cottonwood Falls.

To the House of Delegates:

The Committee on Necrology submits the following list of members of the Kansas Medical Society whose deaths have been reported since the last meeting of the House of Delegates:

Name and Address	*Age*	*Date 1953*
Dr. Charles Wesley Beasley, Lyndon	82	April 7
Dr. Middleton Lee Perry, Topeka	84	April 10
Dr. Anson Sidney Lutgen, Colby	75	April 7
Dr. Ellis Austin Evans, Conway Springs	76	May 30
Dr. Louie Frank Barney, Kansas City	76	June 1
Dr. William Ebenezer Currie, Sterling	81	June 5
Dr. Charles Mooney Fitzpatrick, Salina	74	July 10
Dr. Clyde O. Merideth, Jr., Emporia	46	July 26
Dr. William Redmon Frisbey, Delia	80	July 28
Dr. Robert Lewis McAllister, Marysville	80	Nov. 1
Dr. Orrin C. Lowe, Paola	70	Nov. 3
Dr. Charles Frederick Menninger, Topeka	91	Nov. 28
Dr. Edward Jacob Haerle, Minneapolis	65	Dec. 29
		1954
Dr. Charles Moses Siever, Topeka	79	Jan. 15
Dr. James Albert Settle, Hutchinson	84	Jan. 23

Respectfully submitted,

J. F. Gsell, M.D., *Chairman*

Nominating

L. S. Nelson, Salina, Chr.; C. H. Benage, Pittsburg; O. W. Davidson, Kansas City; J. L. Lattimore, Topeka; N. E. Melencamp, Dodge City.

In accordance with provisions of the Constitution and By-Laws, the Nominating Committee of the Kansas Medical Society announces here the names of candidates who will be listed on the ballot for the annual election to be held on May 6, 1954. Additional nominations, of course, may be made from the floor.

Because some of the members of the Kansas Medical Society might not be personally acquainted with each of the physicians nominated for office, the committee submits the following brief data.

For President-Elect

Dr. John M. Porter, Concordia, has served as secretary and second vice-president and is currently first vice-president of the Kansas Medical Society. He has also been delegate to the American Medical Association and president of the Kansas Heart Society.

For First Vice-President

Dr. Clyde W. Miller, Wichita, has been a councilor and is currently second vice-president of the Kansas Medical Society. He was chairman of the Committee on Public Relations and was the first president of the Kansas Academy of General Practice.

For Second Vice-President

Dr. Conrad M. Barnes, Seneca, besides numerous offices in his local society, has been a member of the Dean's Advisory Committee for the School of Medicine at the University of Kansas and has been president of Kansas Blue Shield.

Dr. Louie J. Beyer, Lyons, has long been an active participant in Kansas politics and has served as a national committeeman from this state. Besides offices in his local society, he has served six years on the Council of the Kansas Medical Society.

Dr. Thomas P. Butcher, Emporia, has held numer-

for a known response

Fully evaluated clinically

...and known as an agent of choice in the treatment of infections caused by gram-positive and gram-negative pathogens, spirochetes, rickettsiae, certain large viruses and protozoa.

True broad-spectrum action

"Terramycin appears to have [a] most widespread effective range...."[1]

Rapidly absorbed

"Terramycin administered orally was absorbed rapidly with the appearance of antibacterial activity in the serum within 1 hour of administration."[2]

Promptly effective

Many "patients...improved rapidly"[3] with return of temperature to normal often noted within twenty-four hours.

Well tolerated

"Terramycin is generally well tolerated...."[4]

Terramycin®

Brand of oxytetracycline

1. Cowart, E. C., Jr.: Mississippi Doctor 29:278 (April) 1952.
2. Sayer, R. J., et al.: Am. J. M Sc. 221:256 (March) 1951.
3. Knight, V.: New York State J. Med. 50:2173 (Sept. 15) 1950.
4. Trafton, H. M., and Lind, H. E.: J. Urol. 69:315 (Feb.) 1953.

Pfizer

PFIZER LABORATORIES, Brooklyn 6, N. Y.
Division, Chas. Pfizer & Co., Inc.

ous offices in his county society, including its presidency. He has been chairman of the Kansas Medical Society Committee on Allied Groups and has served on other committees.

Dr. Dwight Lawson, Topeka, besides numerous offices in his county medical society, has served as president of Kansas Blue Shield and is a member of the Editorial Board.

Dr. John W. Randell, Marysville, besides numerous offices in his county society, has been a member of the Council of the Kansas Medical Society and for the past several years has been chairman of the Committee on Endowment.

For Constitutional Secretary

Dr. James A. Butin, Chanute, has held numerous offices in his county medical society and has been active in the work of the district society of the nine-county area comprising the third councilor district.

Dr. Karl E. Voldeng, Wellington, besides numerous offices in his county society, has been chairman of the Committee on Control of Cancer and is now president of the Kansas Division of the American Cancer Society.

For Treasurer

Dr. John L. Lattimore, Topeka, has been an active participant in Kansas politics, having served four terms in the legislature. He has been president of the Kansas Medical Society and is currently treasurer.

For A.M.A. Delegate 1955-1956

Dr. Laurence S. Nelson, Salina, has been president of the Kansas Medical Society and is currently delegate to the American Medical Association. He has also served the state as a member of the Kansas State Board of Health.

For A.M.A. Alternate 1955-1956

Dr. Lucien R. Pyle, Topeka, has been editor of the Journal and was formerly chairman of the Volunteer Advisory Committee for Kansas. He is currently president of this Society.

Dr. Frederick E. Wrightman, Sabetha, besides serving his county society in various capacities, has been a member of the Legislative Committee and has served as a Kansas representative of the A.M.A. Committee on Legislation.

Respectfully submitted,
L. S. Nelson, M.D., *Chairman*

Pathology

J. L. Lattimore, Topeka, Chr.; T. R. Hamilton, Kansas City; N. P. Sherwood, Lawrence; B. E. Stofer, Wichita; R. E. Stowell, Kansas City.

To the House of Delegates:

Only two propositions have been presented to this committee for consideration.

The first is consideration of an A.M.A. resolution recognizing pathology, radiology, anesthesiology and physiatry as branches of the practice of medicine. This committee strongly approves of this resolution.

The second is consideration of the Morgan Resolution, requesting subsidy at the rate of $25 per autopsy, this money to come from governmental sources. This committee does not approve this resolution, feeling that such a subsidy would not result in better or more autopsies.

Respectfully submitted,
J. L. Lattimore, M.D., *Chairman*

Postgraduate Study

H. H. Jones, Winfield, Chr.; W. H. Algie, Kansas City; M. H. Delp, Kansas City; H. L. Hiebert, Topeka; E. L. Mills, Wichita; H. P. Palmer, Scott City; Maurice Snyder, Salina.

To the House of Delegates:

For the fifth consecutive year, enrollment has shown an increase over the preceding year. It is the committee's belief that this type of education has been established. The subject matter of the program is arranged in conjunction with an advisory group from the medical school after the matriculants to the circuit course have made known their desires. The same procedure will be followed for the remainder of this year.

Respectfully submitted,
Harold H. Jones, M.D., *Chairman*

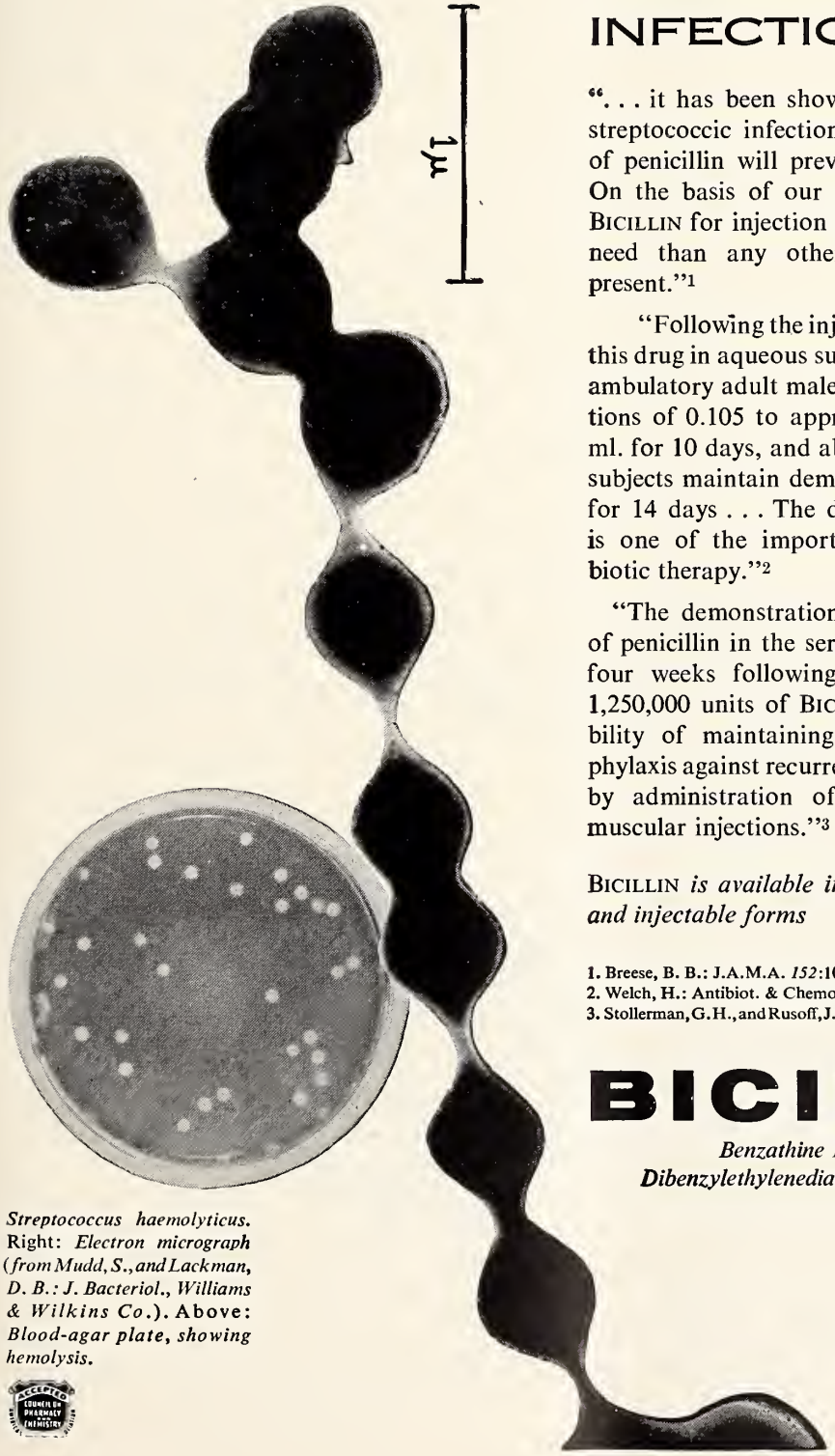
Public Relations

C. W. Miller, Wichita, Chr.; L. G. Heins, Abilene; Robert O'Neill, Topeka; A. J. Rettenmaier, Kansas City; L. F. Schmaus, Iola; C. O. Stensaas, Arkansas City.

To the House of Delegates:

One year ago your chairman made a lengthy report to the House of Delegates concerning the results of an opinion survey and obtained the approval of this body for implementing a program on public relations. This has been attempted during the past year, but, except for a few local enthusiastic responses, the committee regrets to report that very little has been accomplished.

SUSTAINED PENICILLIN LEVELS IN STREPTOCOCCAL INFECTIONS



Streptococcus haemolyticus. Right: *Electron micrograph (from Mudd, S., and Lackman, D. B.: J. Bacteriol., Williams & Wilkins Co.).* Above: *Blood-agar plate, showing hemolysis.*

"... it has been shown that the treatment of streptococcic infections by adequate amounts of penicillin will prevent rheumatic fever ... On the basis of our experience, we feel that BICILLIN for injection more nearly supplies the need than any other product available at present."[1]

"Following the injection of 600,000 units of this drug in aqueous suspension, 100 per cent of ambulatory adult males show blood concentrations of 0.105 to approximately 0.03 unit per ml. for 10 days, and about 50 per cent of these subjects maintain demonstrable concentrations for 14 days ... The development of BICILLIN is one of the important milestones in antibiotic therapy."[2]

"The demonstration of detectable amounts of penicillin in the serum of most patients for four weeks following the administration of 1,250,000 units of BICILLIN suggests the feasibility of maintaining continuous drug prophylaxis against recurrences [of rheumatic fever] by administration of single monthly intramuscular injections."[3]

BICILLIN *is available in oral suspension, tablet, and injectable forms*

1. Breese, B. B.: J.A.M.A. *152*:10 (May 2) 1953
2. Welch, H.: Antibiot. & Chemo. *3*:347 (April) 1953
3. Stollerman, G. H., and Rusoff, J. H.: J.A.M.A. *150*:1571 (Dec. 20) 1952

BICILLIN®

Benzathine Penicillin G
Dibenzylethylenediamine Dipenicillin G

Wyeth®
Philadelphia 2, Pa.

Letters have been written to all county medical societies on at least two occasions during the past year, requesting co-operation in this program. An outline was prepared recommending to county societies the basis upon which mediation committees might be organized. It was strongly urged that this should be made a reality so that no citizen of this state could justifiably state that he had nowhere to turn to correct what he believed to be a grievance against the medical service available to him.

Eleven of 72 county medical societies have responded.

A second suggestion made twice to all county medical societies was the request that some plan be prepared to give each community continuous 24-hour medical service. It is well recognized that the majority of the communities in Kansas have already established this portion of the program and that medical care is available on a continuous basis. It may also well be that they have publicized this fact sufficiently so that all persons in the community are aware of the service. However, five out of 72 county medical societies replied during the year, declaring this portion of the program to be in operation.

A third request was twice submitted to county societies concerning a recommended outline for the establishment of better relations between the radio, the press, and the local medical profession. Three societies have reported activity in that field.

Your committee has available to it hundreds of suggestions from numerous sources, including the public relations department of the American Medical Association, concerning successful projects that have been attempted elsewhere in this field. Also available are numerous intriguing ideas that have been tried by societies within this state. Your committee would be pleased to serve as a clearing house for such ideas and distribute them to the county medical societies, but hesitated because of the apparent lack of interest in many societies. It is the opinion of this committee that a person or a committee should be named in each society to direct the local public relations effort, but, failing up to the present at least, to receive the names of such persons or committees, your Committee on Public Relations has lacked the necessary point of contact.

In spite of the apparent pessimism of this report, your chairman is not at all discouraged. It takes time to create interest in extracurricular activities such as those recommended by this committee. A certain experimental period always precedes enthusiastic support of ventures of this type. Your chairman merely wanted to acquaint the House of Delegates with the present situation in the hope that this body will renew its effort in the direction of enlarging the scope of public relations in this state.

Your committee has dreamed of statewide projects directly in behalf of the Kansas Medical Society. Some, such as the presentation of a network radio or several individual television programs, are currently being explored. These, however, even though time will be contributed by the stations involved, will entail expense and are not recommended for immediate adoption. The immediate need is for the creation of services that will require the expenditure only of time and effort. It is the hope of your committee that these should, under all circumstances, precede the other. So, for the above reasons, it is respectfully suggested that this body find some means for creating a local interest in public relations activity in each county medical society during the coming year. The committee again pledges its continued interest in assisting county societies and looks forward toward the coming year as a period of enlarged accomplishments.

Respectfully submitted,

C. W. MILLER, M.D., *Chairman*

RURAL HEALTH

J. G. Claypool, Howard, Chr.; C. M. Barnes, Seneca; P. L. Beiderwell, Belleville; J. R. Bradley, Greensburg; V. E. Brown, Sabetha; C. S. Fleckenstein, Onaga; E. T. Gertson, Atwood; K. R. Hunter, Lebo; J. J. Marchbanks, Oakley; John Neuenschwander, Hoxie; H. S. O'Donnell, Ellsworth; E. F. Steichen, Lenora; R. P. Stoffer, Halstead; H. J. Williams, Osage City.

To the House of Delegates:

The Committee on Rural Health has had four meetings during the past year.

The committee is continuing its study on the effects of carbon monoxide and tractor vibrations on the health of the farmer. Correspondence with automobile manufacturers, farm organizations, farm equipment manufacturers, the Greyhound Bus Line, and universities has indicated a need for further study, which this committee wishes to continue during the coming year.

Hospital construction in rural areas was discussed at length, and the committee now awaits further developments from a hospital survey which is being made before taking further action.

Mr. Aubrey Gates, field director of the Committee on Rural Health of the American Medical Association, attended one of the meetings and complimented Kansas on the work of this committee. He said Kansas is leading the nation in its rural health program, and he further indicated that many states have implemented the Kansas Plan to help relieve medical prob-

Nationally advertised Surgical Supplies and Equipment have been placed at Topeka, Joplin, Kansas City and St. Joseph for your convenience by—

GOETZE NIEMER COMPANY

Management by Dr. W. F. Goetze, member of the American Medical Ass'n, assures intelligent servicing of your orders.

PATRONIZE JOURNAL ADVERTISERS

It is advertisers who make possible the publication of the Journal in its present form.

They are deserving of your patronage.



lems in rural areas. He felt this committee might well explore ways of implementing the "last mile" in their program on rural health in an effort to provide education for the public in helping them to realize their responsibility in the community. The difference between medical care and community responsibility must be stressed.

The committee approved a motion to appoint a sub-committee to join with a sub-committee of the Committee on Hospital Survey to act as liaison between the hospitals and the Kansas Medical Society to discuss various hospital problems.

The final meeting was held at the University of Kansas School of Medicine in the office of the dean. Members of the Committee on Medical Schools of the Kansas Medical Society met with this group and the dean and department heads to discuss problems in rural health relating to the university. The committee suggested that a preceptor who is a member of the Committee on Rural Health be appointed to the university's preceptorship committee to coordinate the work of both units. This suggestion was accepted with enthusiasm.

It was also suggested that more emphasis be placed on clinical medicine and physical diagnosis in the curriculum of the university. University officials explained that this matter has already been taken care of.

In regard to another question relating to a course in general practice at the university, it was pointed out that such a program is now being conducted.

The committee reported the extreme shortage of nurses in rural areas. In answer, the university officials said that they are now 40 per cent under their quota of registered nurses at the university hospital. It was agreed that this is a national problem which will need considerable study.

There was a long discussion of the preceptorship program in which the Committee on Rural Health suggested that the student would derive the most benefit if he began his preceptorship after all formal courses at the university have been completed, prior to his internship. Because school terms are worked out on a quarterly basis, this proposal would be impossible. The committee also felt that an ideal period for preceptorship would be to train with two different doctors for a period of six weeks each. An eight-week course is the minimum time in which a student can actually benefit from this type of program. It was pointed out that students spend six weeks with a preceptor and six weeks at a state hospital at the present time. It is impossible now to extend the period from six to eight weeks, but this will be kept in mind for the future.

In addition to these meetings, the chairman attended the Rural Health Conference in Dallas, March 4-6, 1954.

Respectfully submitted,
J. G. CLAYPOOL, M.D., *Chairman*

STORMONT MEDICAL LIBRARY

John Crary, Topeka, Chr.; G. F. Corrigan, Wichita; Rodger Moon, Prairie Village; A. K. Owen, Topeka; W. L. Valk, Kansas City.

To the House of Delegates:

This committee has met but has no suggestions as to the management of the library or with regard to any measures of which the library is acutely in need.

It is suggested that further binding of journals be pursued, as in the past. In the future there will undoubtedly be a need for additional filing space for journals.

Respectfully submitted,
JOHN E. CRARY, M.D., *Chairman*

STUDY OF HEART DISEASE

P. W. Morgan, Emporia, Chr.; D. R. Bedford, Topeka; P. M. Clark, Independence; C. W. Erickson, Pittsburg; L. H. Leger, Kansas City; G. L. Norris, Winfield; L. O. E. Peckenschneider, Halstead; E. J. Teeter, Goodland; D. C. Wakeman, Topeka.

To the House of Delegates:

Your Committee on the Study of Heart Disease reviewed the value of mass chest-x-rays and concluded that because so few children under 16 are included in the surveys, this is not of great value in the study of heart disease.

It was decided to prepare an article for the JOURNAL on standard nomenclature in heart conditions. The purpose is to set up wider use of accepted criteria for classification of heart diseases.

A speakers' bureau was endorsed, and it was felt that in order to have this of value it would be necessary to have the close co-operation of the Kansas Heart Association, the Kansas Medical Society, and the Kansas State Board of Health. Both doctors and lay persons will be included in the list of speakers, and the Kansas Heart Association will serve as liaison for this project.

The annual four-day refresher meeting was held at Emporia late in October, and Dr. George C. Griffith of the University of Southern California was guest instructor. The course, as in previous years, was oversubscribed with representation from all areas of the state.

It's Convenient to Buy Kanox

THERAPEUTIC OXYGEN AND ANESTHETIC GASES



Oxygen Therapy
Regulators and Equipment
Repair Service for
Regulators, Flowmeters
and Humidifiers

Nitrous Oxide
Ethylene
Carbon Dioxide
Helium
Oxygen
Cyclopropane

KANSAS OXYGEN, Inc.

Phone 5-5551 HUTCHINSON, KANSAS Box 551



Woodcroft Hospital--Pueblo, Colorado

A private hospital for the scientific treatment of neuro-psychiatric disorders, including alcoholism and drug addiction. Beautiful landscaping and home-like surroundings afford a restful atmosphere. Accommodations vary from single rooms with or without bath to rooms en suite, allowing for segregation of guests.

Detailed information furnished on request.

KARL J. WAGGENER, M.D.

A letter from the committee was sent to the Kansas State Board of Health, asking for co-operation in financing the one-day course in heart disease at the University of Kansas School of Medicine.

The committee welcomes the continuation of the *Heart Bulletin* and the co-operation of the Kansas State Board of Health in this regard.

Respectfully submitted,

P. W. MORGAN, M.D., *Chairman*

VENEREAL DISEASE

G. S. Voorhees, Leavenworth, Chr.; M. L. Bauman, Wichita; J. K. L. Choy, Topeka; H. E. Neptune, Salina; V. M. Winkle, Topeka.

To the House of Delegates:

There is no work specifically assigned to this committee since various agencies and groups in the state are engaged in the control of venereal disease. Consequently, no meeting of the committee was held.

Respectfully sumbitted,

G. S. VOORHEES, M.D., *Chairman*

ANNOUNCEMENTS

Qualifying examinations for fellowship in the International College of Surgeons will be held on May 3 and 4. Written examinations will be given at Cook County Graduate School of Medicine, Chicago, May 3, and clinical examinations will be held at Cook County Hospital, May 4.

The International College of Surgeons also announces a regional meeting to be held at Salt Lake City, July 22 and 23, under the sponsorship of the Rocky Mountain Division. All members of the medical profession are invited.

The Harvard School of Public Health will give postgraduate scholarships in amounts ranging up to $5,000 to qualified candidates during the academic year 1954-1955. Deadline for filing applications is April 30. Inquiries may be addressed to the secretary of the school, 55 Shattuck Street, Boston 15, Massachusetts.

The Medical Library Association will hold its 53rd annual meeting, June 15-18, in Washington, D. C. Headquarters will be the Hotel Statler, and the official host the Armed Forces Medical Library. Information may be obtained from Lt. Col. Frank B. Rogers, Armed Forces Medical Library, 7th Street and Independence Avenue, S.W., Washington 25, D. C.

The American Congress of Physical Medicine and Rehabilitation, 30 North Michigan Avenue, Chicago, announces its annual essay contest. The closing date is June 1. Contributions will be accepted from medical students, interns, residents, and graduate students. The winner will receive a cash award of $200. a gold medal, and a certificate of award.

THERAPY OF TUBERCULOSIS

Because of the problem of drug resistance, the suggestion that streptomycin and isoniazid should not be used together in the treatment of tuberculosis is made in a progress report of the U. S. Public Health Service Co-operative Investigation of Antimicrobial Therapy of Tuberculosis.

"When all regimens tested show equal therapeutic effectiveness," the report states, "differences in the frequency of toxic drug reactions assume increased importance in choosing therapy. In these preliminary findings, isoniazid given in doses of 3 milligrams per kilogram (of body weight) daily produced practically no toxicity, but one of every 10 patients who began treatment with isoniazid at the 10 mg. per kg. dosage developed toxic symptoms which necessitated a change in therapy.

"If confirmed, these findings point to three conclusions: first, that isoniazid, streptomycin, and PAS should not be used simultaneously; second, that, over all, the 3 mg. per kg. daily dose of isoniazid is preferable to the 10 mg. per kg. dose since it equals the higher dose in therapeutic effectiveness and is appreciably less toxic, and, third, the wisdom of wide use of the combination of isoniazid plus streptomycin may be questioned since isoniazid plus PAS is equally effective."

Although it is apparent that interest in group practice has been on the upswing in the United States since World War II, little information is available on the subject. The A.M.A. recently authorized a study of the entire question of group practice, to be undertaken jointly by the Council on Medical Service and the American Association of Medical Clinics.

The proposed field project, employing personal interviews, will seek to answer questions concerning the organization and operation of group practice clinics. A tentative date in early fall has been set for completion of the project.

THE LATTIMORE-FINK LABORATORIES

Topeka — El Dorado
Kansas

J. L. Lattimore, A.B., M.D., Pathologist
A. A. Fink, A.B., M.D., Pathologist
H. C. Ebendorf, M.T., Serologist
A. C. Keith, B.S., Chemist
L. W. Hull, A.B., Bacteriologist
Walter Norris, A.B., Chemist

Anatomical and Clinical Pathology

Containers Furnished Upon Request

Medical and Surgical Supplies for Doctors of Medicine and Hospitals

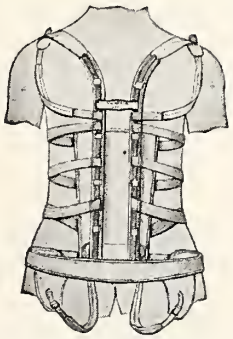
Munns Medical Supply Co.

512 Kansas Avenue
Topeka, Kansas

Deformity Appliances of Quality

Orthopedic and Surgical Appliances

Artificial Limbs
Trusses
Abdominal Supports
Elastic Hosiery
Foot Supports
Surgical Corsets



Taylor Back Brace
Made to Order in
Our Own Factory

P. W. HANICKE MFG. CO.
1009 McGee St. VIctor 4750
KANSAS CITY, MO.

THE MEDICAL PROTECTIVE COMPANY
FORT WAYNE, INDIANA

PROFESSIONAL PROTECTION EXCLUSIVELY SINCE 1899

specialized service assures *"know-how"*

TOPEKA Office:
J. E. McCurdy, Rep.,
1035 Randolph Avenue,
Telephone 2-3027

New Scientific Media Established

New horizons in the field of undergraduate and postgraduate medical education were opened recently when the California Medical Association voted to establish a subsidiary, non-profit corporation, called the Audio-Digest Foundation.

Through a system of tape recordings and synchronized visual slides or film strips, current medical literature and lectures will be summarized and distributed to doctors throughout the world. A board of editors will be established. This advisory group will consist of leading physicians representing all specialty groups. Three types of service will make Audio-Digest of equal value to the specialist, the general practitioner, and the medical student.

These services will be:

1. A weekly, one-hour tape summarizing the current medical literature (approximately 600 journals) from the standpoint of significance and practical usage. This is designed primarily for the general practitioner and covers all fields of medicine. The tape summary was started as an experiment in March of this year and current subscriptions are going all over the world. This material is distributed on a subscription basis. Starting January 15, a bi-weekly, one-hour tape was made available for surgeons. Starting February 15, a similar service for specialists in internal medicine was provided. March 15 was the beginning date for a tape-digest for obstetricians and gynecologists.

A new technique has been developed which makes it possible to make taped literature of lectures available in any language.

2. A complete medical lecture library is being established. This has been available on a limited basis since March. Material is accumulated from on-the-spot recordings at medical conventions, and specially prepared lectures for the Audio-Digest library. This material is either sold or rented.

3. The California Medical Association will begin immediately to assemble "master" lecture tapes from the leading medical school professors in the nation's 79 medical schools. These tapes, covering the entire field of undergraduate education, will be made available to medical school libraries to supplement local lectures.

All lecture material will be reviewed each six months to be kept up to date medically.

Both the taped literature digests and medical lectures can be duplicated and ready for distribution in less than 24 hours after the "master" tape has been made. Profits accruing from the Audio-Digest Foundation will be earmarked for the nation's medical schools.

Major General Silas B. Hays, Army Medical Corps, has approved a pilot plan using this media in both the United States and overseas medical-military installations.

Dr. Sidney J. Shipman, San Francisco, chairman of the California Medical Association's Council, commented: "By making medical literature and lectures available to the world's physicians in their own language and in this new, dramatic form, we hope to contribute something to medical education."

He concluded: "This will be a special boon to the doctor practicing in rural or isolated areas because it will take the profession's outstanding teachers to him when it is impossible for him to go to the medical center to hear the professor. This means that the rural doctor can keep abreast of medicine's rapid scientific advances and at the same time, continue home care for his patients."

Patronize Journal advertisers.

CLASSIFIED ADVERTISEMENTS

FOR SALE OR RENT—Fully equipped 3-room office, ground floor, instruments and medicines. Town of 800. Nearest competition 20 and 35 miles. Write the Journal 4-54.

FOR SALE—Office equipment, Picker x-ray, steel examining room set, examination chair, cabinets, lamps, fans, Alpine lamp, heat lamp, diathermy, maple reception furniture, oak office furniture, safe, instruments, microscope, complete well equipped office. Reasonable price. Optional—will rent eight-room brick office or sell on reasonable terms. Good location, county seat, large territory, first class city-owned hospital, surgeon needed, good will free. Retiring. Write the Journal 5-54.

LOCATION WANTED. Will be released from service April 15, will begin residency October 1. Will assist or relieve physician during May, June, July, August. Formerly in general practice. K.U. graduate, member of Kansas Medical Society. Write the Journal 6-54.

FOR SALE—Portable x-ray (Profex), also permanent standard. Other equipment, all in good condition. Write the Journal 7-54.

PHYSICIAN leaving in August or before for one-year residency wants replacement. Small community. Home and office available. Office equipped with EKG, x-ray, all other necessities. Write the Journal 8-54.

EXCELLENT OPPORTUNITY for man out of internship to gain broad experience with specialist backing. Our group needs a man on a one or two year basis at $1,000 per month. Write the Journal 9-54.

LOCATION—Town of 9,000, 2 colleges, hospital, not overcrowded with doctors, all swell fellows. Modern ground floor office, not much to sell. Partnership if desired. Give qualifications and personal data. Write the Journal 10-54.





BENADRYL®

Patients troubled by lacrimation, nasal discharge, and sneezing respond to BENADRYL and enjoy symptom-free days and restful nights.

MEDICAMENTA VERA
PD & CO.
Parke, Davis + Company
DETROIT, MICHIGAN

TABLE OF CONTENTS

MAY, 1954

ORIGINAL ARTICLES

Complete Heart Block with Morgagni-Adams-Stokes Syncope Treated with Large Doses of Epinephrine: A Case Report—William E. Larsen, M.D., Kansas City, Kansas . . . 249

The Art of Medicine and the Cancer Patient—Ralph E. Jordan, M.D., Holton, Kansas . 252

A Case of Actinomycosis of Ovary—Lyman C. Murphy, M.D., Wichita, Kansas 254

Guillain-Barre Syndrome: Report of Two Cases Treated with Vitamin B Complex, Cortisone, and ACTH for Relief of Pain—Thomas L. Foster, M.D., and Charles Pokorny, M.D., Halstead, Kansas 256

EDITORIALS

Veterans' Affairs 261

Orgone Energy 262

Honor in the Practice of Medicine 262

MISCELLANEOUS

President's Page 260

Clinicopathological Conference 264

Treatment of Hepatic Insufficiency—Senior Thesis 278

Entered as second-class matter at the post office at Fulton, Missouri, June 19, 1953, under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of October 3, 1917, authorized on July 2, 1918. Office of Publication: 1201-05 Bluff Street, Fulton, Missouri.

THE JOURNAL OF THE KANSAS MEDICAL SOCIETY

Editorial Board

ORVILLE R. CLARK, M.D., *Editor,* Topeka

JOHN W. CAVANAUGH, M.D., Topeka

DAVID E. GRAY, M.D., Topeka

RICHARD GREER, M.D., Topeka

DWIGHT LAWSON, M.D., Topeka

Associate Editors

DONALD P. TREES, M.D., Wichita

GLEN R. SHEPHERD, M.D., Kansas City

Information

Ownership: The Journal is a non-profit publication owned and published monthly by the Kansas Medical Society.

Subscription: A year's subscription to the Journal is included in membership in the Kansas Medical Society, with $2.00 of each member's dues apportioned to the Journal. Rates to others, except in foreign countries, $4.00 per year or 60c per copy.

Material: Scientific articles, editorials, and data of general interest are invited from all members. Articles are to be submitted on condition that they are contributed solely to this publication. A right is reserved to reject any material deemed unsatisfactory.

Manuscripts: Only manuscripts that are typewritten on one side, double spaced, and original copies can be accepted. Manuscripts will be returned upon request.

Advertising: All advertising contracts, and all copy from advertisers under contract are subject to approval of the editorial board. Copy should be received by the 20th of the month immediately preceding the month of publication.

Non-Responsibility: Although an effort is made to publish only accurate articles and legitimate advertisements, the Journal denies legal responsibility for any statements, opinions, or advertisements appearing under the names of contributors or concerns.

PAULINE FARRELL, Managing Editor and Advertising Manager

OLIVER E. EBEL, Business Manager

THE KANSAS MEDICAL SOCIETY

President.....LUCIEN R. PYLE, M.D., Topeka

Past President....WARREN F. BERNSTORF, M.D., Winfield

President-Elect.....MURRAY C. EDDY, M.D., Hays

First Vice-President..JOHN M. PORTER, M.D., Concordia

Second Vice-President..CLYDE W. MILLER, M.D., Wichita

Constitutional Secretary..DALE D. VERMILLION, M.D., Goodland

Treasurer.....JOHN L. LATTIMORE, M.D., Topeka

AMA Delegate 1953-1954, LAURENCE S. NELSON, M.D., Salina

AMA Delegate 1954-1955, GEORGE F. GSELL, M.D., Wichita

Councilors and Councilor Districts

First District.....W. L. ANDERSON, M.D., Atchison

Second District .....H. P. JONES, M.D., Lawrence

Third District....CHARLES E. VESTLE, M.D., Humboldt

Fourth District.....F. C. TAGGART, M.D., Topeka

Fifth District.....HAROLD M. GLOVER, M.D., Newton

Sixth District.....NORTON L. FRANCIS, M.D., Wichita

Seventh District..SEVERT A. ANDERSON, M.D., Clay Center

Eighth District.....A. E. RUEB, M.D., Salina

Ninth District.....JAMES L. JENSON, M.D., Colby

Tenth District.....L. W. REYNOLDS, M.D., Hays

Eleventh District.....C. V. BLACK, M.D., Pratt

Twelfth District.....R. G. KLEIN, M.D., Dodge City

OLIVER E. EBEL, Executive Secretary

RUEBEN M. DALBEC, Executive Assistant

Executive Office: 315 West Fourth Street, Topeka

THE JOURNAL
of the
KANSAS MEDICAL SOCIETY
Copyright, 1954, by The Kansas Medical Society

Volume LV MAY, 1954 No. 5

Complete Heart Block with Morgagni-Adams-Stokes Syncope Treated with Large Doses of Epinephrine: A Case Report

William E. Larsen, M.D.
Kansas City, Kansas

The purpose of this paper is to illustrate the dosage level of epinephrine sometimes needed in the therapy of Adams-Stokes syncope of ventricular standstill origin complicating complete heart block.

CASE REPORT

A 48-year-old white man entered the hospital because of frequent "black-out" spells. He was perfectly well until five days prior to admission, at which time he noted the onset of a sore throat, malaise, and a low anterior pain traversing the chest. These symptoms followed an exposure to inclement weather. The following day the patient had one transient episode of fainting while lying in bed. His condition was considered to be due to "viral flu," and he was given antibiotics. From that time until his admission to the hospital, he had fever, profuse diaphoresis, anorexia, lethargy, and a low anterior chest pain which, on the day prior to admission to the hospital, radiated into his left shoulder. On the morning of admission he had three fainting spells of short duration, and for five hours preceding admission he had frequent tonic and clonic convulsive episodes associated with periods of unconsciousness.

The past history was non-contributory to his present illness, except that when he was in the military service in 1940 he was told that he had a slow heart rate. The patient's family and social history were not unusual.

The physical examination upon admission revealed an acutely ill, white man of 48 years of age. His temperature was 100.2 degrees F., his pulse rate was 56 per minute, and his respiratory rate was 16 per minute. Initial observation revealed the patient to be well developed and well nourished, and lying in bed with frequent episodes of syncope accompanied by tonic and clonic convulsions. He was extremely apprehensive. The convulsive episodes occurred at about five- to ten-minute intervals and were of 45 to 60 seconds duration. The lung fields were clear to percussion, and there were fine moist crackling rales heard at both bases posteriorly. The heart rate was 56 per minute and regular. The first mitral tone varied in intensity from beat to beat, and no murmurs were heard.[5] The pulse and heart sounds became imperceptible about five seconds prior to the episodes of syncope. The remainder of the examination was not unusual.

Laboratory examination showed a white blood count of 19,800 per cu. mm. of which 80 per cent were neutrophiles, 12 per cent were monocytes and 7 per cent were lymphocytes. The red blood count was 4.26 million per cu. mm. with 12 grams of hemoglobin. The erythrocyte sedimentation rate was

This paper, submitted for the March (University of Kansas School of Medicine) issue, was omitted then because of lack of space. Dr. Larsen is from the Departments of Medicine, the Veterans Administration Hospital, Kansas City, Missouri, and the University of Kansas Medical Center, Kansas City, Kansas.

42 mm. in one hour. The blood serology was negative, and the urinalysis was normal. A portable chest x-ray revealed an increased density in the lower left chest. A repeat chest x-ray on the seventh hospital day showed normal lung fields. An electrocardiogram taken shortly after admission (Figures 1 and 2)

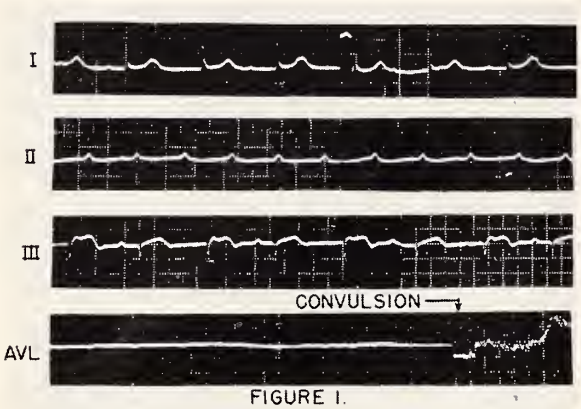


FIGURE 1.

Lead I & III Complete auriculo-ventricular dissociation.
Lead II Auricular complexes with ventricular standstill.
Lead AVL Auricular complexes with ventricular standstill leading to a convulsion.

Electrocardiogram taken on the first hospital day showing complete auriculo-ventricular dissociation and ventricular standstill.

showed a slow, regular rhythm with a complete dissociation between the auricular and ventricular complexes indicating a third degree heart block. In Figure 1 there are only auricular complexes seen in lead II and aVL. There is an elevation of the RS-T segment in leads II, III and aVF with a depression of the RS-T segment in lead V-2. The interpretation is a third degree heart block with periods of ventricular standstill and evidence of an early posterior myocardial infarction.

The patient was placed on therapy of epinephrine, 1:1000 dilution, 0.3 cc. subcutaneously every hour. Because of continued episodes of ventricular standstill and convulsions, it was soon evident that more frequent and larger doses of epinephrine would be necessary. Consequently, the dose was increased to 0.4 cc. subcutaneously every 30 minutes, and ephedrine sulfate 45 mg. was given orally every four hours. Only at this dosage level was the patient free of syncopal attacks.

By the third hospital day, the patient had been free of syncope for 24 hours. The dosage of epinephrine was gradually reduced and was discontinued on the fourth hospital day. With this amount of epinephrine, which totaled 20 cc. of 1:1000 dilution in one 24-hour period and 47 cc. over a period of three days, the patient did not become excited and agitated but had a relaxed feeling as well as relief from apprehension.

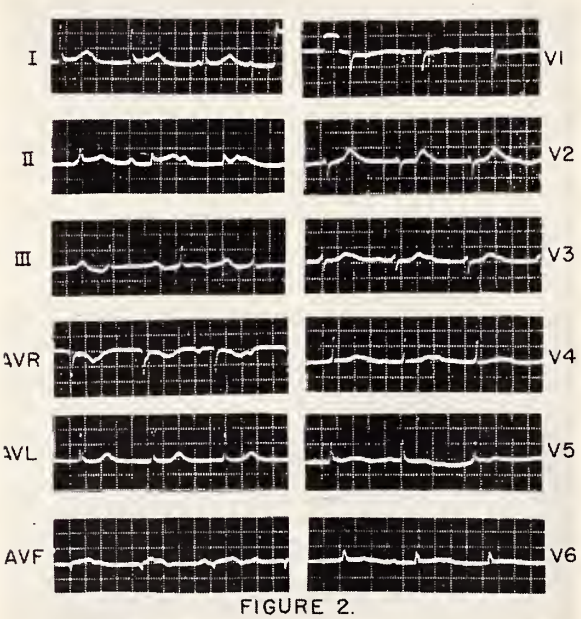


FIGURE 2.

Electrocardiogram taken on the first hospital day showing a complete auriculo-ventricular dissociation.

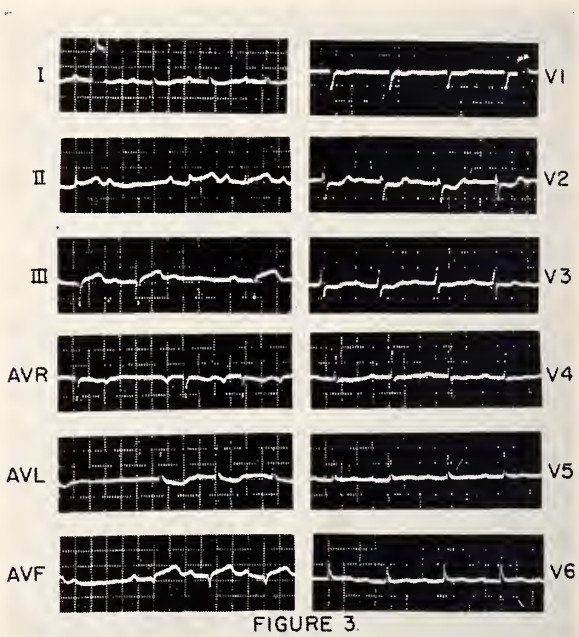


FIGURE 3.

Electrocardiogram taken on the third hospital day showing a second degree heart block with a Wenckebach phenomenon.

Serial electrocardiograms (Figures 3, 4, and 5) revealed gradual recovery from the complete heart block through second and first degree heart block to a normal sinus rhythm. In Figure 3, it will be noted that a Wenckebach phenomenon is present. The persistence of RS-T changes in lead II, III and aVF confirmed the original impression of a posterior myocardial infarction.

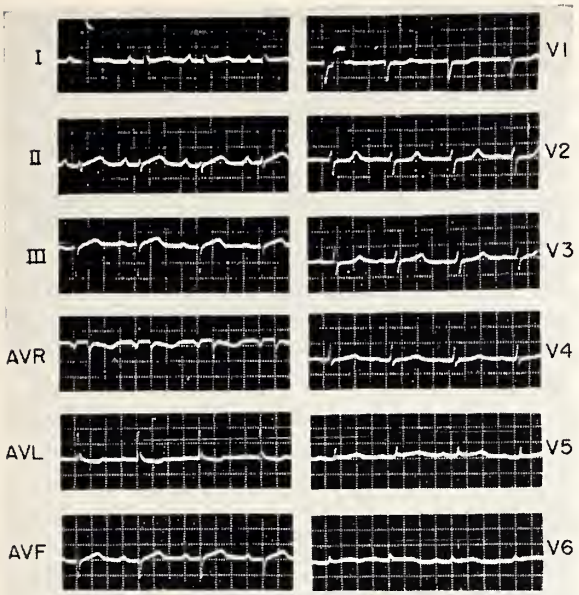


FIGURE 4.

Electrocardiogram taken on the sixth hospital day showing a first degree heart block and evidence of a posterior myocardial infarction.

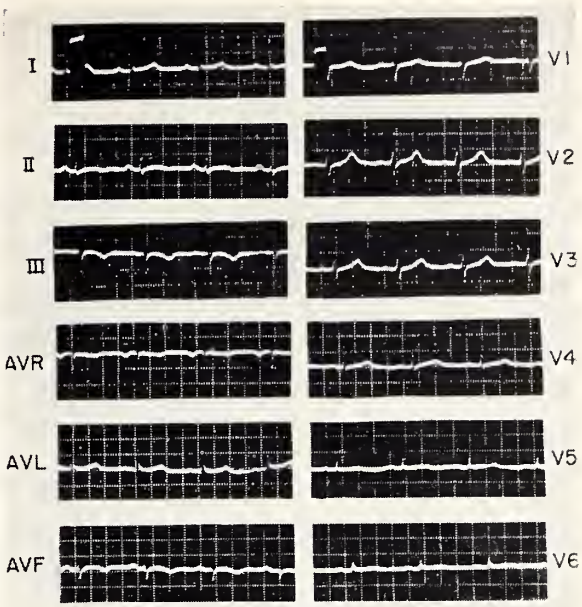


FIGURE 5.

Electrocardiogram taken on the 12th hospital day showing normal A-V conduction and a posterior myocardial infarction.

The progress of the patient was satisfactory, and he was discharged on his 21st hospital day to continue his convalescence at home. A follow-up examination eight months after his acute illness revealed an apparently healthy man with electrocardiographic evidence of myocardial insufficiency and an old posterior myocardial infarction.

DISCUSSION

The rhythm of the ventricular muscle is normally influenced in three manners: (1) via the conductive pathways within the Purkinje system of the heart; (2) by sympathetic fibers via the superior, middle, and inferior cardiac nerves and direct fibers from the upper four and five thoracic ganglia, and (3) by hormonal influence of circulating epinephrine. When the normal pathways are interrupted, an inherent focus in the septal myocardium (A-V node) takes over the function of a rhythmical pacemaker. All myocardial tissue has this inherent potentiality of rhythmicity.

The stimulation of the sympathetic nerves to the heart or an increase in circulating epinephrine to the heart results in (1) an accelerated and more vigorous beat, (2) a shortening of the refractory period as well as shortening of the mechanical and electrical systole, and (3) an increase in the automaticity of all pacemaking tissue. It is this last function of epinephrine which is important in the therapy of ventricular standstill of complete heart block.

When one is confronted with a problem of Morgagni-Adams-Stokes syncope, it is of extreme urgency to determine the cause of the attack associated with an imperceptible pulse because the therapy of ventricular standstill could be disastrous if applied to a case of ventricular tachycardia or fibrillation. The only means of differentiation is the electrocardiographic record during an attack of syncope.

The quantity of epinephrine which was necessary in this case to maintain a regular cardiac rhythm free of asystoles was far in excess of recommended dosages. However, in a case such as this one, to obtain the desired therapeutic effect, the usual dose must be increased several fold. The assay of epinephrine used was verified to be of full potency.[13] During the administration of epinephrine, none of the other effects such as tremor, nervousness, or anxiety was noted.

SUMMARY

A case of Morgagni-Adams-Stokes syncope complicating complete heart block secondary to a myocardial infarction is presented, and the therapy using large doses of epinephrine is reviewed. In one 24-hour period, 20 cc. of 1:1000 epinephrine was needed, and over a period of three days 47 cc. was necessary.

BIBLIOGRAPHY

1. Major, Ralph H.: Classic description of disease, p. 327-339, Chas. C Thomas, Springfield, Ill.

2. Parkinson, J.; Papps, C., and Evans, W.: Electrocardiogram of Stokes Adams attack. Brit. Heart Journal 3:171, 1941.

3. Schnur, S.: Newer Concept of Stokes-Adams syndrome, Medical Record and Annals 44:48-53 (February) 1950.

4. Boer, S. De: On the origin and essence of the Morgagni-Adams-Stokes syndrome, Annals Internal Medicine 37:48-64 (July) 1952.

5. Beard, O. W., and Decherd, G. M., Jr.: Variations in the

first heart sound in complete A-V block, American Heart Journal 34:809-816 (December) 1947.
6. Campbell, M.: Complete heart block, British Heart Journal 6:69-92 (April 10) 1944.
7. White, Paul D.: Heart disease, 4th ed., Macmillan, 1937.
8. Goodman, Louis S., and Gilman, Alfred: Pharmacological basis of therapeutics, Macmillan, 1941.
9. Fulton, John F.: A textbook of physiology, Saunders, 1950.
10. Best, Chas. H., and Taylor, Norman B.: Physiological basis of medical practice, 5th ed., 1950, Williams and Wilkins.
11. Pastor, B. H., and Worrilow, S. H.: Electrocardiographic patterns in Stokes-Adams syndrome, Annals of Internal Medicine 34:80-89 (January) 1951.
12. Phear, A. G., and Parkinson, J.: Adrenaline in the Stokes-Adams syndrome, Lancet 1:1933, 1922.
13. Personal Communication with the Gold Leaf Pharmaceutical Company.

The Art of Medicine and the Cancer Patient

Ralph E. Jordan, M.D.

Holton, Kansas

When a patient is presented with the diagnosis of cancer for which specific measures are not immediately initiated nor advisable, and when this patient returns to his home, the local doctor is confronted with a real problem in philosophy and medical management.

Having been presented with such patients, the writer has evolved several bits of philosophy which are adamant. First, and most prominent, it is felt that a definite prediction as to the time of the patient's death is not within the province of any doctor. The outlining of "time" either to the patient or to the patient's relatives does not seem to be medically correct in that the endurance of that particular individual is an unknown; in that the course of any cancerous cell is not predictable even with histological support; and in that the variables of cancerous cells *in situ* are too numerous to permit accuracy of prediction.

In essence, it is felt that the greatest injustice that a surgeon, a radiologist, or any doctor can do to the patient, to the family, to himself, or to the family doctor, is to make a definite pronouncement regarding the probable time of dying. The patient returns home with a psychotic noose around his throat—the rope being tightly secured—so the family doctor has little hope of overcoming the fear of the predicted time. Any efforts toward encouragement are thwarted by previous and more authoritatively received words.

Every doctor can recall among his patients some one patient who spent several months "dying," only to go on and live for years; in fact, the patient finally and actually dies of "other causes." A specific example—a woman underwent surgery in Topeka in early 1947. She was informed directly and indirectly that six months was the most time possible for a patient with her type of cancer to survive. Therapeutic x-ray was administered, some supportive medications were given, and the patient was requiring paracenteses for abdominal ascites when first seen—two years later—in November 1949.

The patient was seen at her daughter's home in Topeka as a courtesy. The first impression—even in consideration of the ascites—was that the patient was more discouraged than she was terminal in her appearance. In addition, a herpetic lesion over the left eye made life's noises the more annoying to her. Words of encouragement were given. It was implied that she could yet spend Thanksgiving and even Christmas in her own home, and a detailed written program of living, eating, and medication was outlined with a definiteness that re-enforced the idea that it was worth the effort and that "life was expected to continue."

The patient spent that Christmas (1949) in her own home. The paracenteses were less frequently necessary. In fact, the patient and her husband were able to revisit their childhood communities during the next summer. They were able to spend another Christmas (1950) fairly pleasantly and in their own home. The woman quite suddenly became ill in January 1951 and died within a 48-hour period.

It is true that some 120 therapeutic x-ray treatments were given after surgery and before November 1949. It is likewise true that a total of 17 paracenteses were required from November 1949 until January 1951. Conversely, it must be stated that the woman spent 6 months dying, another 12 months wondering why she hadn't, and another 24 months living an apparently well adjusted life—a pleasant woman who resumed her practices of sending candies and treats to friends, neighbors, and children.

Parenthetically it is recorded that the daughter's husband, in whose home the patient spent her first 18 months of illness, packed up and disappeared without leaving any word as to his destination. To my knowledge, he has never returned to his wife and children. The entire story might have read differently if "hope" had been expressed after surgery rather than "time" having been impressed.

Written at the invitation of the Committee on Control of Cancer, Kansas Medical Society.

This paper proposes to outline a practical philosophy toward the management of such problems. It is hoped that patients and relatives have not been too thoroughly "doomed" to be unredeemable.

The philosophy of Sir William Osler in his lecture, "A Way of Life," is appropriate—"to live in day-tight compartments." Or, to quote Carlyle, "Our main business is not to see what lies dimly at a distance, but to do what lies clearly at hand." The future is today! This philosophy can be skillfully injected even into a cancer-minded patient.

The patient's cupboard is too often too full of pills for morning, afternoon, and bedtime; the room is too empty of the elements of encouragement to continue to live for today and each day. A positive attitude toward what to do, what to eat, and where to go is beneficial to the patient, his family, and even to the doctor. The end of any life being unpredictable, energies are better expended on encouragements than upon the "hypos" of discouragement.

Specific areas of preachment are believed to be practical and acceptable to the patient and to the relatives. Thus, it is proposed that a positive preachment of an adequate diet be made; that the seven basic foods be presented; that the value and necessity of regular meal times be stressed; that the food be well prepared, attractively served, and actually eaten.

One is reminded of the story of the political prisoner who dressed formally for each meal, ate of the food served him, and outlasted by some 20 years all of the others who were originally imprisoned with him. It is felt that the time and attention given by the doctor to stressing the necessity of adequate meals, actually eaten, will be repaid by a patient who becomes more co-operative and stays happier longer.

Secondly, there is considerable medical writing concerning the probability of cancer being a "deficiency" disorder. Similarly, there are many articles in the lay press which impart these same thoughts. The pens of authors who are trying to publish medical papers in the lay press and the pens of the various faddist groups continuously promote this thinking. In either case, the positive approach to an adequate diet usually meets with the family's approval and is physically and psychologically desirable for the patient.

I recall a family having several lawyer sons who commented to the doctor, "Now that the scientific measures have been outlined, just what do we feed the man?" It may be a correct accusation—that we are overzealous in our medical management and are unwittingly negligent in simply feeding the patient.

In medical writing, an adequate diet can be advocated by nutritionists as being proper for the so-called healthy adult. Much work is being done on the normal requirements of the geriatric patient. It would seem proper to feed such patients at least a normal diet.

Within the field of patients having Hodgkin's disease, it is reported that Bacillus acidophilus, oral penicillin, and oral sulfathiazole alter the intestinal tract to a situation in which the patient feels better and the usual anemia is more easily controlled. It is not contended that the course of the cancer is altered, but it is noted that the immediate well-being of the patient is improved. Quite often the patient becomes intrigued with dietary possibilities and will read with alertness and real interest every available article. He continues to live.

Other research would hint that the various sources of vitamin B should be abundantly supplied. The drinking of an abundant amount of water by count and not simply by thirst often freshens the patient remarkably.

In short, regardless of the supposed scientific "value-less-ness" of these suggestions, attention to an adequate meal plan, attention to possible favorable products, and attention to rules which should apply to general health all make sense to the patient. Something is being done by him and for him. Ideas have replaced the "emptiness" of awaiting a predicted fate. Foods, vitamins, tonics, and amphetamine buoyants can be used much longer and with much more acceptance than are the usual depressant bromides, barbiturates, and narcotics.

Again, encouragement to live today, and to do today as much of as many things as you can is definitely a form of treatment. It can be commented that it is never safe to be alive; nevertheless, the patient must be encouraged to live. He must be encouraged to continue his vocation and his hobbies. The financial ship should be made more secure and more definite, but it should not be abandoned. There is a great difference between having legal matters in order and the drawing of a "finis" line on the entire endeavor. Similarly, it is better to plant the bush and never see the rose than it is to quit planting.

A by-product of this writing can be the admonishment of the medical doctor to preach encouragement, to predict the possible continuation of living, and to avoid the "emptiness" of looking only to life's termination. A program of "nothingness" is felt by the patient and the family to the extent that they are actually wedged into the care of faddists and cultists who do promise and who do see that something is actively being done.

We have failed as physicians when we forsake a patient who is ill with cancer and when we, by negativism, cause him to turn to other groups for care. So-called modern medicine, together with personalized encouragement, should be superior to the too-often administered colonic irrigations.

Throughout this writing there are no new suggestions as to specific therapy. It was assumed that the diagnosis implied failure of our scentific endeavors. Contrariwise, there is the definite thought that we should be wary of suggesting a time of life's termination to any and every such patient, but rather that we should adopt and positively present an active program to these patients, that a po outlook on each day of life is desirable and po that enforcement of an adequate diet is favoral ceived by the patient, and that a period of rela satisfactory living can be inserted between the of diagnosis and the time that final supportive are becomes necessary.

A Case of Actinomycosis of Ovary

Lyman C. Murphy, M.D.
Wichita, Kansas

It has been aptly said that, in the practice of medicine, it would be better not to diagnose the rare and unusual until the common well known ailments can be ruled out—also that the only excuse for publishing a scientific article is to add to, or simplify, preexisting knowledge. However, the rare and unusual promptly finds its place in the literature when it does occur, and the following presentation of a case of ovarian actinomycosis is along this line.

Actinomycosis is a disease caused by a fungus which grossly appears in the form of "sulfur granules" lying in small pockets of pus; microscopically it consists of club shaped filaments arranged radially like the sun's rays, hence the name "ray fungus."

Grossly, its lesions are a suppurative granulomatous process more commonly seen in cattle, located at the angle of the jaw and popularly known as "lumpy jaw." The earliest report of the disease was in 1845. Transmission from cattle to human beings has not been proved. Most of the human cases occur in persons who have had no contact with diseased animals.

It has been suggested that minor traumatism, as from fragments of grain imbedded in the soft tissue of the mouth, may be a factor in the infection.

In the earlier reports it was considered that the source of infection might be *exogenous* or *endogenous,* in the former being due to chewing grasses, grains, or cornstalks. In the latter it is believed that the fungus is a "normal inhabitant" of the alimentary tract, where, aided by minor trauma and perhaps by secondary invaders in the form of cocci or bacilli, it may attack the human body. Since the ray fungus is an obligate anaerobe, the exogenous theory of infection is untenable. However, another species of the Actinomyces group called "Nocardia" is an aerobe and causes lesions similar to Actinomyces bovis. Its lesions are called "nocardiosis," said to begin most often in the lungs but spreading to other parts of the body.

From this, one might get the reaction that "a rose by any other name is just as sweet," or postulate a theory of one's own that the ray fungus is a "Dr. Jekyll and Mr. Hyde" type of organism living first as a facultative anaerobe, an innocuous exogenous existence under an assumed name (Nocardia), and becoming dangerous only as an obligate anaerobic organism (Actinomyces bovis) under favorable conditions as previously noted.

From its primary location in one part of the body, it is believed that the organism may be spread to other organs by contact or through the blood stream. Some believe that the primary source of pelvic lesions may be the vagina (use of pessary or abortion followed by infection). Some cases have even been believed to be primary in the pelvis. The pelvic organs, when invaded, consist of ovaries and tubes with occasional involvement of parametrium. When only one ovary is affected, the right is reported as being most often involved. (Previous operation such as appendectomy followed by adhesions is found in many cases.) Involvement of pelvic organs occurs chiefly in women before the menopause. Characteristic laboratory findings: r.b.c. normal, mild hypochromic anemia, elevated w.b.c., sedimentation rate increased, urine apt to contain pus. Diagnosis (in suspected cases) is confirmed by microscopic examination, cultures, and microscopic examination of tissues.

Mrs. O. H., housewife, age 37, consulted her physician July 25, 1953, complaining of pain in epigastric region, accompanied by chills, fever, and abdominal distention. She had had morning sickness for three weeks and thought she might be pregnant. (Frog and rabbit tests produced prompt death of the animals.) She had lost 30 pounds in weight, unaccountably.

Physical examination revealed abdominal distention and a mass in the pelvis which was sore and tender. The impression was pelvic inflammatory disease, probably salpingitis.

Vaginal examination August 1, 1953: Cervix long,

xed and open, no mass nor tenderness on right eft parametrium tender, and suggestion of mass uterus.

eration (10 days after onset): Mass involving tubes, ovaries, and parametrium, filled with one cu of white odorless pus. The patient was given lots of antibiotics and bled profusely during the operation, requiring several blood transfusions.

She left the hospital on the 13th postoperative day and had some elevation of temperature one day afterward. She was still draining on August 21, 1953, but had no elevation of temperature, and antibiotics were stopped.

Summary of pathologist's report (specimens hardened in formalin): Specimens consist of two ovaries and tubes. The larger (left) ovary is 4 x 4 x 2½ cm. containing a previously opened 2 cm. cavity at one pole suggestive of abscess. Much of the adjacent ovarian tissue is pale yellowish-white and on gross section shows numerous tiny orange-colored granules imbedded in its substance, but no grossly visible pus. An occasional follicle cyst is seen in the cortex. The corresponding tube is 1¼ cm. thick along its distal third. The ostium is patent.

The right ovary is slightly smaller with congested surface and contains multiple follicle cysts (up to 4 mm.). The corresponding tube is 1 cm. thick with patent ostium (Figure 1).

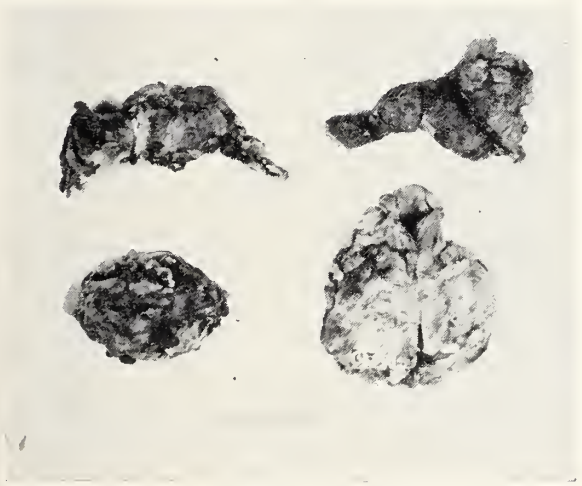


Figure 1

Microscopic sections of the left ovary show, in addition to wall of abscess noted grossly, numerous ray fungi surrounded by pus cells (Figure 2).

The opposite ovary shows mild active inflammation. No fungi are found in this ovary.

Sections of the tubes show swollen mucosal folds thickly infiltrated by neutrophiles and plasma cells. The lumens contain purulent exudate. No fungi are seen in the sections examined.

Diagnosis: actinomycosis of left ovary; active inflammation (mild) of right cystic ovary; bilateral pyosalpinx.

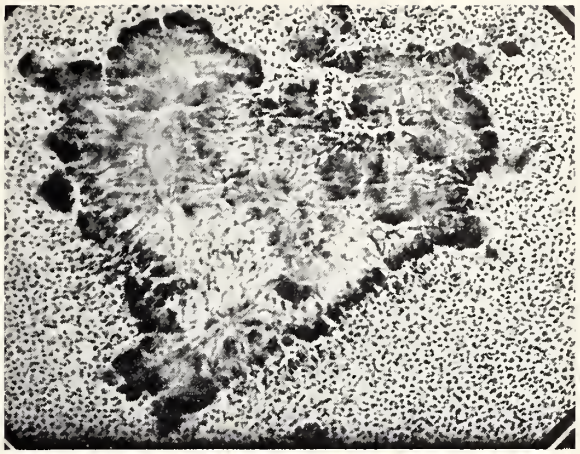


Figure 2

The patient was later admitted to the Mayo Clinic, and she was again operated upon. Details are unknown at present. She was dismissed about the last of October and returned home, reported to still have a draining sinus from the pelvis through the skin. She was advised to return to the Mayo Clinic after a period of time for a check-up.

Comment: A rare case of human actinomycosis involving only the left ovary, with bilateral pyosalpinx and mild active inflammation of the opposite ovary, is presented. The case should be of interest to surgeons and gynecologists. The disease is particularly to be suspected in cases with a history similar to that presented, when the condition does not yield readily to antibiotics and is followed by persistent draining sinuses following operation. The ray fungus is hard to culture, rendering tests for sensitivity to various antibiotics difficult. Sulfadiazine and penicillin seem to be the ones most used.

Prior to the advent of antibiotics and chemotherapy, KI was the chief therapeutic agent. It is reported that with antibiotics the prognosis is better, but actinomycosis is considered a serious condition with high mortality.

According to Herbut, slightly over 100 cases of actinomycosis of tubes and ovaries had been reported by 1949. He states that the intestinal tract is probably the primary site of infection.

Numerous details regarding acid fast characteristics of the fungi, characteristic clinical laboratory findings including increased sedimentation rate, etc., have been omitted in this presentation, but they may be found in the previous literature on this subject.

Much appreciation is due Dr. W. W. Weltmer of Beloit, Kansas, and Dr. C. A. Hellwig of the Hertzler Clinic for their help in preparing this article.

BIBLIOGRAPHY

Putnam, H. C.; Dockerty, and Waugh: Actinomycosis, Surgery 28:5, 781-800 (Nov.) 1950.

Lisa, J. R., and Levine, J.: Actinomycosis of tubes and ovaries, Arch. Path. 23:53-7 (Jan.) 1937.
Campbell, D. A., and Bradford, B.: Actinomycosis of thorax and abdomen, Arch. Surg. 57:202-16 (Aug.) 1948.
Nichols, D. R., and Herrell, W. E.: Penicillin in treatment of actinomycosis, J. Lab. & Clin. Med. 33:521-525 (May) 1948.
Paalman, R. J.; Dockerty, M. B., and Mussey, Robt. D.: Actinomycosis of ovaries and fallopian tubes, Am. J. Obst. & Gynec. 58:3, 419-31 (Sept.) 1949.
Rashbaum, M., and McIntosh, Harriet: Pelvic actinomycosis treated by surgery and roentgen ray, with recurrence, J. Obst. & Gynec. 17:849-859 (June) 1944.
Neiman, B. H., and Fahrner, A. H.: Actinomycosis of ovary, Am. J. Obst. & Gynec. 45:534-8 (March) 1943.
Bubis, J. L.: Two cases pelvic actinomycosis representing hard and soft types, Medical Times 73:8:227-234 (Aug.) 1945. (Mt. Sinai Hosp., Cleveland, O.)
Singleton, Bruce: Penicillin in treatment of case of "pelvic" actinomycosis, East African M. J. 27:5, 208-210 (May) 1950.
Paton, A.: Pelvic actinomycosis (abridged version of the Sutton Sams Price essay), St. Thomas' Hosp. Gazette 45:2, 42-48 (April) 1947.
Ingalls, Edgar G., and Merendino, K. Alvin: Actinomycosis of female genitalia, case report and review of literature, West Surg., Obst. and Gynec. 60:9:476-479 (Sept.) 1952.
Baker, Everett M.: Actinomycosis of internal female genitalia, ibid. 55:9, 501-507 (Sept.) 1947.

Guillain-Barre Syndrome: Report of Two Cases Treated with Vitamin B Complex, Cortisone, and ACTH for Relief of Pain

Thomas L. Foster, M.D., and Charles Pokorny, M.D.
Halstead, Kansas

A number of excellent reviews of the literature on Guillain-Barré syndrome have been published by Hand and Rudoy,[5] MacNeal and Bland,[8] and Stillman and Ganong[10] within the last few years. Pathological reports by DiFiore[2] and Lowenberg and Foster[7] quite adequately explain its protean clinical manifestations on the basis of distribution of the pathological lesions.

Although this condition shows many of the characteristics of a specific disease entity, we must consider it a syndrome until more definite causative factors can be determined.

Prior to Guillain, Barré, and Strohl's[4] first paper in 1916, many cases had been reported under such headings as infectious neuritis, infectious polyneuritis, radiculitis, acute ascending myelitis, Landry's paralysis, myeloradiculitis, neuritis, neuronitis, and many other terms. It remained for the above authors to call attention to the characteristically high spinal fluid protein with a normal or at least relatively low cell count.

The onset of this condition is usually fairly acute and frequently follows an upper respiratory infection. However, in this clinic we have had one case that occurred shortly after a severe gastric hemorrhage and another that terminated fatally following a gallbladder operation. Most patients give the history of a fairly acute onset beginning with decrease in motor function and sensory loss accompanied by paresthesia. Muscle pain may be present in varying amounts. In one of our patients, constant severe back pain and numbness of the fingers were present two weeks before weakness was observed.

DiFiore reported a series of cases which well demonstrates the variable clinical picture. In his series he reports one patient with a monoplegic involvement, another who was paraplegic, an ascending type of onset, and a generalized type (with or without facial diplegia). The onset is usually accompanied by a low grade fever and very little change in the blood count, and it apparently runs a self-limited course which is, of course, suggestive of an infectious disease.

When death occurs, it is usually due to respiratory paralysis. Consequently, one should always have a respirator readily accessible to the patient. Once the patient gets over the acute phase, convalescence is usually continuous although frequently prolonged, due to the length of time required for regeneration of the long nerve fibers. Residual paralysis or permanent damage has been reported in some cases, but frequently recovery is complete.

The two cases we are reporting were of the generalized type with sixth nerve weakness and severe pain. Both cases have made a complete recovery with no residual changes. However, they did have more pain than is usually present. In one patient the pain was limited to the back, while the other patient complained not only of severe back pain, which was continuous, but of a severe, sharp, stabbing pain in all extremities. In fact, it was this patient's severe pain that led us to consider the use of ACTH.

As stated above, the cause of Guillain-Barré syndrome is not known. Gavin[3] and Kennedy[6] have sug-

From the Departments of Neuropsycbiatry and Internal Medicine, Hertzler Clinic and Halstead Hospital, Halstead, Kansas.
Read before the Mid-Continent Neuro-Psychiatric Society in Kansas City, September 26, 1953.

gested that it could be an acute infectious process. In that case, ACTH or cortisone would certainly be contraindicated; on the other hand, Reitmen and Rothschild[9] believe that the condition is the result of an allergic phenomenon, and of course in such a case it would be the treatment of choice. A review of the literature revealed one case successfully treated by Stillman and Ganong.

CASE 1

Mr. W. B., a 43-year-old white married farmer, was referred to the Halstead Hospital by Doctors D. A. Hardman and L. W. Bauer of Smith Center, Kansas, on February 11, 1952. He came with the complaints of numbness in the fingers and toes, fatigue, irritability and pains in the back and all over the body, with onset two to three weeks previous.

Present Illness: The patient had not slept well the past two months, but his appetite had been good. At Christmas time, six weeks before admission, an incident occurred in this man's personal life which he found upsetting and frustrating at that time and about which he worried. However, at the onset of this condition he felt that he had come to accept the matter and was taking it in his stride.

Past History: The patient had no emotional upsets or serious illnesses in the past. He had sciatica a few years before which was quite painful, but he had had no trouble for several years. The family history was negative.

At the time of the physical examination, the patient was mentally dull, probably due to recent medication; however, he was constantly twisting and turning in bed, trying to find a more comfortable position. He complained of weakness and pain in the legs a week prior to admission and described this as a severe, constant type of pain. He also complained of sudden, sharp, shooting pains in all his extremities but principally in the right leg. Examination of the patient on admission revealed a well developed, well nourished, rather muscular man. The neck was stiff. The straight legs could not be elevated more than 35 degrees. The patient exhibited a great deal of soreness in the muscles of the calves of both legs and in the right thigh and back. Pupils were equal, regular and reacted to light and accommodation; extraocular movements were normal. There was no complete paralysis of any muscles; the grips of both hands were weak; he showed considerable weakness in his legs, could barely lift the heel off the bed; biceps, Achilles, and knee jerks were markedly decreased; there was a suggestive Babinski's sign on the right; abdominal and cremasteric reflexes were absent. Temperature was 98°, pulse 70, blood pressure 130/80; heart, lungs, and abdomen were negative.

Laboratory Findings: Urine negative. Sedimentation rate 20 mm. in the hour. Hemoglobin 84 per cent, rbc. 4,300,000, wbc. 11,300, eosinophiles 2, juveniles 2, stabs 19, segmented 52, lymphocytes 24, and monocytes 1 per cent. Fasting blood sugar was 105, blood chloride 612, N.P.N. 35.2, creatinine 167, urea nitrogen 15.0 mg. per 100 cc. of blood. Spinal fluid: color clear, pressure 14 cm., polys 2, lymphocytes 1, protein 252 mg., the gold curve 1111232110. The Wassermann was negative.

Course in the Hospital: During the first 24 hours the patient had a thoroughly miserable time in spite of frequent injections of codeine and demerol; 100 mg. of demerol would give him partial relief for approximately 2 hours, when he would again complain of severe pain. On the third hospital day the patient was started on ACTH, 10 mg. in 5 per cent glucose solution, in a very slow drip. We attempted to give this over a 20-hour period. This was repeated daily for 3 days, then it was reduced to 7.5 mg., 5 mg., and 2½ mg. respectively for the next three days, at which time it was discontinued and cortone was started, 25 mg. four times a day by mouth.

Within a few hours after administration of ACTH, the pain was markedly relieved, and the patient was comfortable with only 100 mg. of demerol in each 24-hour period. During the fifth day, when the patient received only 2½ mg. of ACTH, and on the following day when he was on cortone, his pain again became severe. ACTH was begun again, 10 mg. daily, and was continued for the next five days, reducing the dose to 5 mg. after the third day. On the sixth day in the hospital, three days after ACTH was started, the patient developed a total paralysis of the right sixth nerve and experienced some respiratory difficulty.

On the 10th day in the hospital, the patient developed a rather typical toxic psychosis. He became confused, disoriented, delusional, and hallucinated. His nights were restless, he would constantly call for the family, he would think the hospital on fire one minute and a little later that some other catastrophe was threatening.

ACTH was discontinued on the 13th hospital day, and the patient was started on cortisone, 25 mg. q.i.d.; three days later this was reduced to 25 mg. t.i.d. and gradually reduced to 25 mg. b.i.d. Following the discontinuance of ACTH the second time, the psychosis cleared up almost immediately, and the patient experienced little pain. Within a few days he was able to be out of bed and to move around the room. The patient left the hospital on the 33rd day, able to walk short distances. The diplopia had completely disappeared, and, aside from showing extreme fatiguability and weakness, he was comfortable. In spite of the clinical improvement, the spinal fluid protein (252 on admission), which had dropped to 196 on the 11th day, was up to 270 mg. on the 23rd day—10 days before dismissal.

Six months later this man was examined in the clinic and showed no changes in sensation or reflexes. He was still complaining of some weakness. He stated that he could work in the field running a tractor quite comfortably for half a day but became very tired if he attempted to work longer.

CASE 2

Mrs. F. W., a 52-year-old housewife, was referred to this hospital on December 3, 1952, by Dr. A. C. Eitzen of Hillsboro, Kansas. The patient's chief complaint was numbness and weakness in all four extremities, with onset approximately 5 weeks before admission. She had been doing considerable work, redecorating her house, reaching under cabinets, and climbing on ladders. Following this, she noticed some stiffness and soreness in her back between the shoulders but ascribed it to the exercise.

About three weeks prior to admission, while she was lying in bed, her feet felt "like there were big crusts on the bottom." This numbness gradually increased until she had little feeling in her feet, and the same symptoms started in her hands. She also developed a marked weakness in her feet and legs and was unable to stand alone. About ten days prior to admission, she developed a diplopia on looking to the right. Dr. Eitzen reported, the day before her admission to the Halstead Hospital, that the spinal fluid protein was 98, cell count 0, Wassermann negative.

The past and family history were consistently negative. She had always been healthy, had had four normal deliveries and no serious illness or surgery.

Physical examination revealed a rather obese white woman, apparently clearly oriented and free of any mental symptoms. Blood pressure was 120/80, pulse 80, temperature normal; heart, lungs, and abdomen were negative. Pupils were equal, regular, and reacted to light and accommodation, and the fundi were negative. There was a definite weakness of the sixth nerve on the right. Facial muscles were normal, the tongue was normal, speech clear. There was generalized weakness of all four extremities; abdominal reflexes were absent. The patient was barely able to lift her heels off of the bed and was totally unable to stand. She could not feel or identify objects held in her hand and recognized only rather deep pin pricks over the hands and feet as painful. She did complain bitterly of considerable pain throughout the entire region of the thoracic spine and the lower cervical region. This was not aggravated by movement, and she insisted that she had not been relieved by analgesics.

Laboratory Findings: Urine was negative. On admission hemoglobin was 76 per cent or 13 gm., rbc. 3,760,000, wbc. 12,000, eosinophiles, 2, stabs 8, segmented 55, lymphocytes 33, monocytes 2, pathological 0. Liver function as determined by the hippuric acid test was 97 per cent. Kline was negative. Spinal fluid: lymphocytes 4, polys 0, protein 78, gold curve 0112443210. Fasting blood sugar 78 mg., N.P.N. 35.0, creatinine 1.75, urea nitrogen 16.0, chloride 595 mg. per 100 cc. of blood. Sedimentation rate 29 mm. in the hour; 31 mm. 12 days later. Sixteen days after admission, the spinal fluid pressure was 24 cm., lymphocytes 2, polys 0, protein 89 mg. Hemoglobin on the 12th hospital day was 80 per cent or 13.5 gm.

Course in the Hospital: The patient was started on ACTH, 10 mg. in 1000 cc. of 5 per cent glucose, given at a slow rate. This was begun on the second hospital day and continued until the seventh hospital day, when it was decreased to 5 mg. and decreased again to 2½ mg. on the eighth day, after which it was discontinued entirely. The chest x-ray on the 15th hospital day showed some flattening of the left diaphragm with blunting of the costophrenic angle and some tortuosity of the aorta.

For three days after cortone was started this patient continued to get weaker and the sensory loss increased. On the fourth hospital day she was unable to pick up a spoon off her tray to feed herself, but by the eighth hospital day her strength had returned sufficiently so that she could lift her food to her mouth, though she was totally unable to locate objects on her tray by touch. However, during this time the pain was entirely relieved, and she was comfortable. The patient continued to convalesce and left the hospital on the 27th day, barely able to walk to the car.

This patient was seen again one month after dismissal, at which time spinal fluid had a cell count of 10 lymphocytes, 4 polys, protein 35. She is able to do part of her own housework and gets around fairly well.

DISCUSSION

In both of these patients, the acute process of the disease was continuing to progress at the time of admission to the hospital. Both were started on ACTH, which afforded them almost immediate and complete relief from pain. In the first patient, the pain was extremely severe. When ACTH was discontinued and cortisone substituted, his pain recurred. When ACTH was given again until it had to be discontinued because of a toxic psychosis, pain was relieved. In both cases, muscle weakness and sensory loss progressed for at least four days after ACTH was started. Vitamin B complex was given in addition to ACTH and cortisone. The highest spinal fluid protein obtained on one patient was after recovery had begun. Both patients have recovered with no residual symptoms.

Since knowledge of the etiology of this condition is as yet only theoretical, there is no specific treatment. There have been several reports in the literature

of patients treated with ACTH and cortisone; most authors have felt these drugs are of value. Since several of the vitamin B group have proved to be absolutely essential to the nervous system and their absence in itself will produce a nutritional disturbance, practically all physicians give vitamin B and particularly B-12 on an empirical basis. Both of our patients received vitamin B complex by mouth and 30 micrograms of B-12 intramuscularly every third day.

CONCLUSIONS

1. We feel that ACTH was of marked value in offering these two patients complete relief from pain.

2. On the fourth day after treatment with ACTH was begun, both patients began to improve, though previously the disease had been progressing steadily. We have no way of knowing how much the ACTH actually influenced the course of the disease since it is a self-limited illness; we have seen many cases suddenly start improving spontaneously.

3. It is possible that ACTH helped in clearing up both cases with no residual symptoms. However, the convalescent period was approximately the same as in patients we previously treated by other means.

BIBLIOGRAPHY

1. Creaturo, N. E.: Infectious mononucleosis and polyneuritis (Guillain-Barré syndrome), J.A.M.A. 143:234-236 (May 20) 1950.

2. Di Fiore, John A.: The Guillain-Barré syndrome, J. Nerv. & Ment. Dis. 116:146-156 (August) 1952.

3. Garvin, John S.: Infectious mononucleosis with Guillain-Barré syndrome, J.A.M.A. 151:293-294 (January 24) 1953.

4. Guillain, G.; Barré, J. A., and Strohl, A.: Sur un syndrome de radiculonevrite avec hyperalbunose du liquide cephalorachidien sans reaction cellulaire. Remarque sur les caracteres et graphiques des reflexes dendineux, Bull. et mem. de Soc. med. d. hop. d. Paris 40:1462, 1916.

5. Hand, Morton, and Rudoy, Martin: The prognostic significance of the Guillain-Barré syndrome, Ann. Int. Med. 29:91-102 (July) 1948.

6. Kennedy, Foster: Infective neuronitis, Arch. Neurol. and Psych. 2:621, 1919.

7. Lowenberg, K., and Foster, D. B.: Polyradiculoneuritis with albuminocytologic dissociation: Pathonanatomic report of 3 cases, Arch. Neurol. and Psych. 53:185-190 (March) 1945.

8. MacNeal, P. S., and Bland, J. H.: Guillain-Barré syndrome; review of the literature, current implications and report of 7 cases, Am. Pract. Digest of Treat. 1:337-346 (April) 1950.

9. Reitmen, N., and Rothschild, Karl: The non-infectious nature of the Guillain-Barré syndrome with a possible explanation for the albumino-cytologic dissociation, Ann. Int. Med. 32:923-934 (May) 1950.

10. Stillman, J. Sydney, and Ganong, W. F.: Guillain-Barré syndrome: Report of case treated with ACTH and cortisone, New England J. Med. 246:293-296 (February 21) 1952.

Home Accidents Cost Money

The financial cost of medical care required as a result of accidents each year in Kansas is, of course, unknown. Some inkling as to the size of the burden might be gained from the following information obtained from Kansas Blue Shield for a few selected types of claims paid during the year 1952, the majority of which resulted from accidents. The payment figures represent actual amounts paid by Blue Shield and not actual physician charges.

CLAIMS PAYMENTS FOR CHILDREN UNDER 19 DURING 1952
BY KANSAS BLUE SHIELD

Diagnosis	*Number*	*Payment*
Fracture of Radius and Ulna	554	$ 22,649.76
Fracture of Tibia and Fibula	155	7,636.16
Traumatic Wounds	2,634	22,403.70
Burns (Emergency Treatment Only)	122	594.50
Diagnostic X-ray (Accident Only)	4,836	31,857.55
Total		$ 85,141.67

The estimated average number of individuals under 19 years of age covered by Kansas Blue Shield during 1952 was approximately 99,400. This represents about one-sixth of the population of Kansas in this age group. If the childhood population of Kansas not covered by this insurance plan experienced accidental injuries of this degree of severity at a comparable rate, the cost to parents for partial physician fees alone would approximate $520,000 for just these few selected types of non-major injuries in one year.

Committee on Child Welfare

PRESIDENT'S PAGE

DEAR DOCTOR:

This will be necessarily brief as your new president is in a considerable state of confusion. (This may be the understatement of the year.)

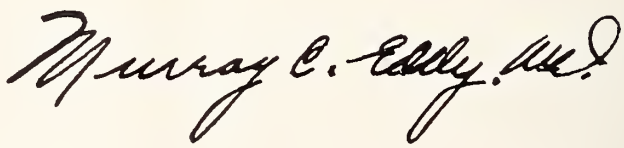
Committee appointments are practically complete, but the year's program can hardly be outlined until after the American Medical Association meeting in June. It is my understanding that several issues should be decided at that meeting which directly affect any legislation we might be considering, and our course cannot be set until later.

I would like to pay a tribute to Dr. Lucien R. Pyle, whose devotion to Kansas medicine, whose calm judgment of controversial issues, have been an inspiration to me and the other officers of the Society. The groundwork done by him and those before him will continue as before to point the way to better medical citizenship, constantly improving medical care for our citizens, and a closer medical community.

I would appreciate personal letters of suggestion from any of you, and I will undoubtedly ask for much advice from all officers, councilors, and committee members as well as the membership at large.

I shall endeavor to be a conscientious servant of the Society.

Yours,



EDITORIAL COMMENT

Veterans' Affairs

At a meeting in Denver on Friday, March 19, representatives of the American Medical Association presented and reaffirmed their stand on medical care for veterans. These representatives made it clear that the A.M.A. places discharged veterans in two separate classes. First are those with service connected disabilities, and second are those with non-service connected disabilities.

Service connected disabilities are those incurred or aggravated in military service, or those suffered following military discharge which are a result of or attributable to military service. Non-service connected disabilities are those ailments which in no way relate to the veteran's service in the armed forces.

The A.M.A. makes it clear that they are in favor of having the federal government provide every reasonable benefit to veterans who were disabled as a result of their military service. On the other hand, they believe that the veteran who suffered no physical or mental mishap while in military service should obtain and provide for his own medical care as does any other individual.

Since this is the stand of the medical profession, the A.M.A. believes we must continue not only to answer questions in regard to non-service connected cases, but we must repeatedly confirm our stand on the government's responsibility to those with service connected disabilities, as it is at this point where many fail to understand our policy. The A.M.A. is in accord with the provision that the government take care of service connected disabilities, and it has cooperated and will continue to assist in providing such care.

For the purpose of this summation, however, let us consider those who are non-service connected cases or those who, when discharged from service, were in a state of physical and mental health as good as or better than when they went in.

A brief history of the medical care program describes the tremendous growth of the Veterans Administration program to veterans with non-service connected ailments. In 1926, just two years after veterans with non-service ailments were receiving treatment for diseases or injuries not incurred in service, 17 per cent were in this class. Two years later, 49 per cent of admissions were non-service connected. In 1931, the non-service cases accounted for 71 per cent of the admissions. By 1944, the sum of $218,000,000 had been spent for veterans' hospital construction, and during that time 80 per cent of admissions were treated for conditions not related to military service.

The Veterans Administration's own figures for the fiscal year 1951 show about 85 per cent of the patients were treated for non-service connected disability, which means 85 per cent of the more than half a million patients.

It is obvious, therefore, that we have had more than adequate facilities to take care of veterans with service connected disabilities for some time, which makes it equally obvious that much of the amount already spent for veterans' care, as well as anticipated expenditures, is for non-service connected cases. There is little need of mentioning that both the number and percentage of non-service connected cases rise steadily. Consider the impact of the basic trend by realizing that by the year 1970 there will be more than three times as many living veterans over 45 years of age as there are today and more than 10 times as many living veterans over 65.

The Veterans Administration now has a total of almost 120,000 hospital beds and is spending more than $600,000,000 annually on its program. In 1945, General Frank T. Hines, administrator of Veterans Affairs, said that by 1975 a fair estimate of hospital beds for veterans would be 400,000 with an annual expenditure of $1,000,000,000.

The A.M.A. urges that the federal government discontinue the care of non-service connected cases except for war veterans with tuberculosis and those with mental or nervous disorders who are unable to pay for their own hospital care, and that even this temporary exception be made until local facilities are adequate to handle such cases.

The country must decide the basic question of whether the cost of the present and future program is a justifiable burden on the taxpayers of this country. The consideration must be studied with concern for the entire population and not a particular segment.

There are many reasons for the A.M.A. stand on this subject. The artificial shortage of doctors created by heavy demands on the medical profession by the government creates a public relations problem sometimes difficult to explain. This same demand also creates shortages in allied personnel, which necessarily means higher wages to attract nurses, technicians, etc., to civilian hospitals. This naturally creates higher prices for the civilian consumer of hospitalization.

The average hospital stay in a Veterans Administration hospital is 30 days, compared to 7½ days for a person with similar disability in a civilian hospital. Many times this brings the over-all cost of illness higher in a VA hospital than in a civilian hospital, even though the medical care is secured without charge. Loss of time at work and travel expense are among the governing factors in this observation.

Today there are about 20,000 veterans waiting to get into veterans' hospitals, of which only three have service connected disabilities. This substantiates the fact that additional hospitals which are being built are constructed specifically for the purpose of taking care of non-service connected cases. All of these factors and many more are important in considering the over-all picture of veterans' medical service.

The most important question the American people must face today, however, is one of American principle. Should veterans who come out of uniform healthy and unhurt be absolved of all personal responsibility for their own health care? Should non-veterans continue to assume responsibility for their own care in addition to taking on additional tax responsibility to pay for a gigantic veterans' medical care program? The answer is found in studying the basic American principles involved.

Orgone Energy

Have you ever heard of orgone energy? Probably not. But perhaps you have patients who believe that it possesses properties that will prevent and cure a variety of serious diseases, including cancer.

Physicians, of course, can spot a medical "phony" immediately, and they may believe that the lay public is discerning enough to recognize and reject impossible claims. However, with science making daily announcements of new discoveries, new "miracles," it is not surprising that some are deluded by false hopes in health fields. Orgone energy is a case in point.

It was in 1939 that Dr. Wilhelm Reich, who once was associated with Dr. Sigmund Freud in Vienna, entered this country and devoted his energies to the invention and production of an "Orgone Energy Accumulator." It was promoted through pamphlets carrying such titles as *The Discovery of the Orgone, Cosmic Superimposition, People in Trouble,* etc.

The most widely distributed model is a cabinet a little smaller than a telephone booth, constructed of alternate layers of wood, metal, glass, and steel wool, with no electrical or other energy connections. This box is supposed to accumulate the "orgone energy" from the atmosphere. The patient sits in the box, and allegedly stored up energy enters his body and cures his ailments. Other models, based on the same accumulation theory, are the "Shooter Box," "Orgone Energy Blanket," and "Orgone Energy Funnel."

You think no one would be gullible enough to place confidence in such a device? The Food and Drug Administration, United States Department of Health, Education, and Welfare, estimates that more than 1,000 were sold or rented at prices ranging from $15 to $225 each.

No further threat exists as far as orgone energy is concerned. The Food and Drug Administration took action in March to bar such devices from interstate commerce. The decree also bans distribution of claims that orgone energy exists and calls for the destruction of all promotional material containing instructions for the assembly and use of the enjoined devices. But the problem remains—the public must be protected from the perpetrators of such sales.

It is interesting to note the reaction of the discoverer of orgone energy to the injunction. When the action was filed, Dr. Reich published a response in which he stated, "Inquiry in the realm of Basic Natural Law is outside the judicial domain, and I shall not appear in court as the 'defendant' against a plaintiff who by his mere complaint already has shown his ignorance in matters of natural science."

The following comment was made by the Commissioner of Food and Drugs, Charles W. Crawford: "Dr. Reich has long contended that only the hopelessly ignorant could disagree with his theories or doubt his miraculous cures with Orgone Energy Accumulators. Repeated challenges were issued in literature, widely distributed by the Wilhelm Reich Foundation, daring medical researchers and physicists to test accumulators adequately. FDA accepted the challenge and has concluded that there is no such energy as orgone and that Orgone Energy Accumulator devices are worthless in the treatment of any disease or disease condition of man."

This particular issue is unimportant to Kansas physicians. It serves only to stress the fact that the public must be safeguarded in the field of health, where irreparable harm may result to persons who abandon or postpone the usual forms of medical treatment while pinning their faith on worthless devices.

Honor in the Practice of Medicine

Much has been said of late to the effect that the physician of today does not occupy the same position of influence in the community that he occupied a few decades ago, and that he has lost dignity in the estimation of the laity. It has also been claimed that even "modern medicine, with all its advantages, improvements and enlarged resources" stands before the public shorn, apparently, of much of that dignity and appreciation that was formerly extended to a less developed science. That this is true in some measure cannot be denied; but it is doubtless due to the commercial spirit of the age in which we are living. The change in social position is due largely to the changes that have taken place in the people. A half century ago the interests of the country were less diversified, and its wealth less developed and distributed.

Many of the luxurious habits of today were un-

known to public and private life. Then education and character were quite universally regarded as the highest measures of worth, and counted for more than wealth; but today the influence of wealth and the commercial activity of the country seem to overshadow and detract attention from the more quiet channels of life in which the physician, from necessity, must have his sphere of activity.

I do not share the pessimistic view that the profession today, as a whole, is unworthy of the high regard and confidence that was once reposed in it by the public. To the contrary, if education, morality, a high type of citizenship and a sacrifice of self in undeviating devotion to duty were accepted as the measure of true worth, no calling in the land would occupy a more lofty height on the pinnacle of worldly esteem than the medical profession of today. However, there are some among the physicians of today who are not faultless, but whose misguided conduct fails to merit either our indulgence or our approval. It is to be regretted that the tendency on the part of some of the well-meaning members of our profession is to disregard the traditions of the profession and to give themselves over to the commercial spirit of the times which is, alas! too common in many walks of life.

The above is a portion of the president's address made before the Kansas Medical Society by J. E. Sawtell, M.D., Kansas City, Kansas, in May. 1908.

Blue Shield Appointment

The appointment of Thomas R. Reed, Topeka, as physician relations representative for Kansas Blue Shield was announced last month by Proctor Redd, director of professional relations for Blue Cross-Blue Shield. Mr. Reed began his new duties May 1. He has been an enrollment representative of the plans since 1951.

Student Center Opened at K.U.

The new $600,000 student center at the University of Kansas Medical Center was officially opened after dedication ceremonies held on Sunday, April 4. The building houses the Battenfeld Memorial Auditorium and the C. B. Francisco Memorial Lounge.

Among the speakers taking part in the dedication were Dr. W. Clarke Wescoe, Dr. Franklin D. Murphy, and Dr. Galen M. Tice.

The auditorium is the only one at the center that can accommodate the entire student body and faculty simultaneously, 924 seats. It is equipped with theater seats, theater lighting, staging, and projection booth. It was constructed and completely furnished by a gift from the Battenfeld Memorial Fund established by Mrs. Edward H. Hashinger in memory of her son, Dr. Jesse R. Battenfeld, Jr., who died in 1945 in a military plane crash.

The auditorium will be used for student assemblies, postgraduate courses for physicians, student-faculty meetings, and for public information and service programs.

In addition to the lounge, auditorium, and dining room which seats 125 persons, the center also includes a book store for medical texts, a snack bar and a recreational library on the first floor. A seminar room, recreation room, showers, and storage rooms are in the basement. The second floor contains 13 double hotel-type rooms for visiting lecturers and participants in postgraduate courses.

Patronize Journal advertisers.

SERVICE SEPARATIONS

As a service to physicians and communities in this state desiring additional medical personnel, the Journal of the Kansas Medical Society will publish in this column each month the names of medical officers who will shortly be separated from the armed forces. These are men who volunteered from Kansas, and many of them will probably be interested in finding locations in this state. Anyone interested in contacting these physicians may write to the address here given.

Theodore L. Batchelder, M.D.
Garden City, Kansas

Albert I. Decker, Jr., M.D.
826 Alabama Street
Lawrence, Kansas

Lyle Harris Edelblute, M.D.
1210 Thurston Street
Manhattan, Kansas

Wendell L. Good, M.D.
Perry, Kansas

Simon E. Hershorn, M.D.
Wesley Hospital
Wichita, Kansas

J. Gilleran Kendrick, M.D.
4224 East English
Wichita, Kansas

Kenneth L. Knuth, M.D.
Atwood, Kansas

Francis X. Lenski, Jr., M.D.
703 South Cottonwood
Iola, Kansas

Stanley R. McEwen, M.D.
447 North Belmont
Wichita, Kansas

Clinicopathological Conference

CASE PRESENTATION

Dr. Paul Sherman, resident in surgery: This is a 64-year-old white man who was admitted on the surgical service December 4 and died five days later. When he entered, he was not oriented to time, place, or person. He was an obese, hot, and dry male with tachycardia and labored, fast respirations.

The history itself, not obtainable from the patient, was given by his son. Apparently it began November 26, after the Thanksgiving Day dinner. The patient became nauseated and had abdominal distention and discomfort. The symptoms progressed. He was taken to his local hospital where he was treated with gastric suction and intravenous fluids. His symptoms of abdominal distention, fever, and malaise continued and, just prior to his admittance here, he became disoriented.

The past history revealed the patient had always been a healthy, hard working, heavy-eating man who had never before been hospitalized. He had had typhoid fever at age 18 and had had mumps as an adult. He had had an attack similar to his present illness three years ago and was said to have been jaundiced for three weeks. During this illness his weight decreased to 180 pounds but thereafter returned to 225 pounds, his approximate weight at time of admission.

On review of systems it was thought that perhaps he had precordial pain with severe exercise, but this was not clear. Genitourinary history revealed a nocturia of one to two times a night and a decrease in the size of the urinary stream.

The family history revealed that one sister died at age 68 of tuberculosis. One sister died at age 70 of heart disease. His father died of a stroke. His mother had hypertension.

The physical examination revealed a disoriented, elderly, obese white male with florid facies, hot dry skin, and dyspnea. He volunteered no complaints. Temperature was 103.0 F. Pulse was 120 per minute, regular. Respiration was labored, 24 and 28 per minute. Blood pressure was 120/80. Body weight was 224 pounds with height 68 inches. Oral mucous membranes were dry; there was no rigidity of the neck; eye, ear, nose and throat examination was unremarkable.

Examination of the thorax showed a moderately increased AP diameter plus obesity. Respiration was diaphragmatic in character. Percussion notes were resonant throughout; although the diaphragms were high, they were mobile. No rales were heard. The heart sounds were faint without murmurs or thrills. There was a tachycardia. The peripheral vessels did not show undue distention.

The abdomen was obese and moderately distended with moderate tenderness in the epigastrium to deep palpation. No rebound tenderness could be elicited. No bowel sounds were heard; fluid or masses were not palpated in the abdomen. Percussion note was tympanic over the entire abdomen. The rectal examination showed benign prostatic hypertrophy of Grade I to II; otherwise the rectal examination was negative. Extremities were normal. The neurological examination was not significant.

Laboratory examination of the urine showed it to be acid in reaction, 1.022 specific gravity, heavy trace of albumin, some white blood cells, and numerous crystals. Remaining urinalyses during his hospital course were obtained by catheterization and showed many red blood cells, 10 to 20 white blood cells per high power field, one negative urine culture, and on three different occasions a specific gravity of 1.010. The blood studies showed hemoglobin of 14.7 grams (90 per cent); 5.3 million red cells; 27,000 to 34,000 leukocytes with an increase in the polymorphonuclears and from 2 to 21 non-filamented forms in repeated examinations. Wassermann and Kahn were negative. Serum calcium and phosphorus was 3.3 and 5.9 mEq./liter respectively on admission, and 4.6 and 6.5 respectively the day he died.

On December 5, 1953, serum amylase was 64 milligrams per 100 milliliters, and serum lipase drawn the same day and reported on the day of death was 1.22 units. On admission, the NPN was 116 and rose to 138 the day of death. Creatine was 6.7 and reached a maximum of 8.0, December 7, 1953, and 6.0 on the day of death. Blood sugar was 155 on admission. Serum sodium was 151 mEq./l. on admission and 159 the day of death. The CO_2 was 20 mEq./l. On December 7, 1953, the blood pH was 7.37. Urine and gastric suction contents were analyzed for sodium, potassium and chloride to aid in maintaining proper electrolyte balance. Serum agglutinations for typhoid O and H, parathyroid A and B, and Brucella were all negative.

Hepatogram on December 7, 1953, showed a direct bilirubin of 0.7 mg. per 100 ml., a total of 1.0 mg. per 100 ml. Alkaline phosphatase was 1.5 millimol units. The cephalin flocculation was 3 plus. Thymol turbidity was 4, serum albumin 3.0, globulin 2.0, total cholesterol 175 mg. per 100 ml. with 50

From the University of Kansas Medical Center. Edited by Glen R. Shepherd, M.D., and Mahlon Delp, M.D., from recordings of the conference participated in by the departments of medicine, pathology, radiology, and surgery, and the third and fourth year classes of medical students.

per cent cholesterol esters. A blood culture on December 6, 1953, was negative. The report was postmortem.

While in the hospital the patient was treated with intravenous dextrose, normal saline, potassium chloride solutions, Miller-Abbott tube intestinal suction, streptomycin, and penicillin therapy intramuscularly. Special sigmoidoscopy was done immediately upon admission and was found to be negative to 27 centimeters. The medical consultant added to the history that the patient's onset of illness was accompanied by cramping abdominal pain.

The temperature showed a drop to normal during the first 24 hours with an immediate rise again to 103° F. by the end of the first hospital day, and thereafter a gradual decrease to 102° F. throughout his stay. The pulse rate decreased and remained about 90 through this period. Respiratory rate continued to be rapid and labored, 36 to 40 per minute. By the second hospital day, the urine output was 1700 cc. and remained good thereafter.

On December 6, 1953, the patient began to pass gas and some loose watery stools of brown color. On December 7, 1953, he had multiple green stools.

During his hospital course his blood pressure was maintained within normal limits until his death. During that day it had been approximately 140/80 with pulse rate of 96 and agonal temperature of 102.6°.

Dr. Delp, chairman: Are there any questions concerning this history?

Q: Were the stools the last two days still multiple and green?

Dr. Sherman: Yes, small in amount, multiple, green, about 5 to 15 a day.

Q: Do you have any more information about the severity of the pain experienced on Thanksgiving Day?

Dr. Delp: He apparently did not consult a doctor on that day. He didn't consult a doctor until two or three days after that.

Dr. Sherman: That's right, he consulted a doctor several days later at the insistence of his family.

Q: Do you know anything about his past history before this present illness and what was his urinary output during the time that he was in the hospital and preceding the hospitalization?

Dr. Sherman: We're not sure about the past history. Hypertension was suggested during two talks with his son. His urine output was low on the day of admission. It was 500 to 700 cc. With intravenous fluid administration, it became 1700 cc. the second 24-hour period and thereafter about 1700 cc. to 2000 cc. per 24 hours.

Dr. Delp: Any other questions? If not, we have some x-ray films as well as an EKG. Will someone put on the EKG for us?

Mr. John Gorman, fourth year student: The first tracing on this patient was done December 3. Another was done on December 8. This pattern would be that of tachycardia with left ventricular strain. It is also the pattern you see in hypopotassemia, with the lowered T waves, the long swooping RST segments and the increased QT. The December 8 tracing is normal.

Dr. Delp: Relihan, let's see the x-rays.

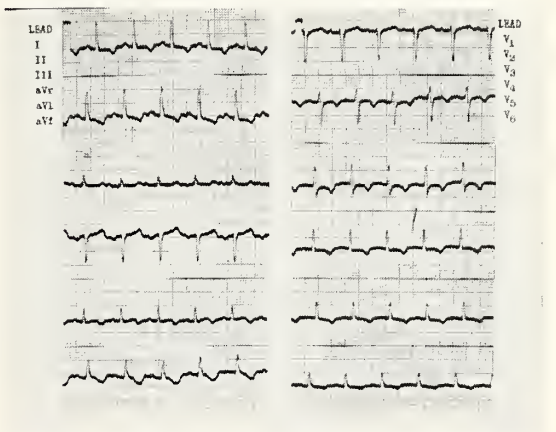


Figure 1. Admission EKG, December 3, showing sinus tachycardia, T waves inverted in leads I, II, aVF, and V_2 through V_6, increased QT and long RST segments indicating left ventricular strain and hypopotassemia.

Mr. Donald A. Relihan, fourth year student: We have two films made on the day of admission, the 4th, one chest film, and one flat plate of the abdomen. The chest film was portable with the patient poorly positioned. Possibly some of our interpretation would be invalidated because of that. The positive findings are elevated diaphragms and soft diffuse infiltrated lung field. I think this possibly could be interpreted as pneumonia.

The flat plate of the abdomen shows dilated gas-distended sections of large and small bowel, with a stepladder pattern of the small bowel in mid-abdomen. Barium enema study on the next day, the 5th, shows dilated proximal portion of the colon. The distal colon is not dilated, possibly due to the fact that he had been previously decompressed by enemas. The widely dilated gas-distended small bowel is still apparent. This is a post-evacuation film which shows retention of the barium in the colon. This pattern is what I would interpret as adynamic ileus without evidence of obstruction at any point, and the pattern of the colon is normal. The next film was made on the 8th, the day before death and in this film we can see the Miller-Abbott tube. There has been some decompression, although it is possible to see the small bowel in some portion of this film still distended by gas.

Dr. Donald R. Germann, assistant professor of radiology: I might add one point. I think the stu-



Figure 2. Flat plate of abdomen made December 4 showing dilated gas-distended sections of large and small bowel with stepladder pattern of the small bowel in mid-abdomen.

dent's interpretation of ileus was good, but one thing that concerned us which didn't come out was that this enema was done during the night. The next morning when we looked at our film we were concerned by certain shadows we saw. It looked like some barium might have leaked out of the sigmoid into the peritoneal space. We were afraid that we had actually perforated the sigmoid. That was the reason for this film which was taken the next morning. There was about 12 hours difference. No evidence of perforation appeared. We were satisfied in our own minds that this was not a perforation, but it was one of the things that went through our minds.

Dr. Delp: Any further questions about the x-rays? They might have some importance to the solution of the problems. It might seem that this is a rather straightforward case, a clinical entity that should be immediately recognized, but I'm sure that that is not so.

These things have interested me. The pain must have been rather mild at onset. At least we can grade it by saying that the patient was not forced to call a physician. It was only after two or three days of illness that he did finally see a physician. He was evidently quite ill then, because hospitalization was recommended immediately and he subsequently was transferred here for further care.

I think we must answer the questions as to whether or not true intestinal obstruction or simply an adynamic ileus, as Relihan has indicated, was present. I would like to hear some explanation for the patient's convulsive disorder which he apparently had the last several days of his illness. The blood chemistry changes also need explanation. Mr. Smith, may we have your differential diagnosis?

DIFFERENTIAL DIAGNOSIS

Mr. Wallace B. Smith, fourth year student: The case for today presents, as Dr. Delp has said, less of a problem right now than it did when I first read the protocol. I think in any discussion we should always try to tie as many things to one disease state or one diagnosis as possible. We should always look for the simplest explanation of a patient's illness.

The patient, if I might review a little bit, had pain in his abdomen. He had nausea, vomiting, and distention. In addition to this, he had persistent fever together with an increasing amount of disorientation. On admission to this hospital, he had a distended abdomen. It was mildly tender, especially in the epigastrium. He had a low, fairly fixed specific gravity urine with some crystalluria and some pyuria. He had a leukocytosis, a low calcium, and a high phosphorus. He had an increased sodium, a decreased chloride, a hyperglycemia and an azotemia. He was treated vigorously with fluids, antibiotics, and other accepted treatments including electrolytes. They were analyzed carefully to determine just exactly what he needed. In spite of this, he continued to worsen and die.

It's a toxic type of manifestation all the way through with a fever and leukocytosis. I think that we are obligated to explain these facts. I think the principal concern we have here, at least when he came into the hospital, is why he was distended and why he had an ileus. I'm not going to go through all the causes that are possible for an intestinal obstruction or an ileus, but I would like to include some of the things suggested by this history.

First of all, the man had a sister who died at age 68 of tuberculosis. Tuberculous enteritis often produces a type of colicky pain with an ileus which may or may not be present. The x-ray picture should show some evidence of ulceration, or we should have some pulmonary signs of tuberculosis or some clinical history suggestive of tuberculous process. We have none here.

Second, it could be neoplastic, either benign or malignant. These things may be asymptomatic until they produce the ileus. However, the x-rays cannot point to anything which we can interpret as a constrictive neoplastic or a space-taking neoplastic lesion.

Third, he could have an obturation of the small bowel, and there are several things that cause this. The only thing I think we need to think of here is possibly a gallstone obturation due to a choledocho-

or cholecysto- duodenal fistula with a possible gallstone in the gastrointestinal tract. The man has a past history of similar abdominal pain with some jaundice. We can assume that the man had cholecystitis or cholelithiasis. He's fat and a heavy eater.

Fourth, the man could have a volvulus. This shows up usually in men past middle age. At least it is seen more frequently in males in late middle life. It frequently follows ingestion of a heavy meal. These people are seized with abdominal pain, usually quite severe, and it's accompanied by more or less distention, depending on where the volvulus occurs. In the sigmoid area, it can produce a great deal of distention. I think the fact he had a sigmoidoscopy shortly after entering the hospital suggests perhaps that was the admitting diagnosis. However, I think that it was pretty well ruled out by 27 cm. of negative sigmoid and no evidence of a volvulus by x-ray.

Another cause of an adynamic ileus that we have to entertain is acute cholecystitis, either by itself or with a possible rupture of the gallbladder, with a bile peritonitis developing. This man had a history suggestive of cholecystitis or cholelithiasis, and I think that we can't rule it out on the basis that we have here. This man had an ileus, and it could have been on the basis of a gallbladder rupture or acute cholecystitis. The pain is not typical, and the man didn't look like he had peritonitis, at least unless he was so foggy that he just couldn't respond at all. I don't believe he was comatose. I think that pneumonia and empyema can cause an ileus with a diaphragmatic pleurisy. Although he had a leukocytosis and a fever, he didn't have the other signs that go with pneumonia. Uremia by itself can cause an ileus. I'm not sure of the mechanism of this ileus, but it has been reported as being seen and not too infrequently in uremia.

A disease that frequently can produce a similar picture to the one we have here is acute pancreatitis. The onset of acute pancreatitis is with sudden severe abdominal pain. It usually radiates to the left back underneath the scapula. The patient usually goes into shock. We don't have that history here.

There is a certain percentage of cases in which this is not seen, and the patient may have just colicky pain. Also we have other evidence of acute pancreatitis, I think. On admission, a low calcium and a high phosphorus were found. It's the sign of pancreatitis with fat necrosis and calcium deposits which show up in the omentum and peritoneum at exploration. Also the loss of calcium soaps through the gastrointestinal tract from a lack of pancreatic enzymes produces a low calcium, and you find a reciprocal high phosphate value. He had a hyperglycemia. This could be accounted for by the destruction of pancreatic tissue.

The amylase and lipase reports that we have are not startling. The amylase was within normal limits, but we would not be concerned about that because we didn't see him when he first had this attack. The amylase could long since have become normal again. The lipase is elevated only slightly, and I think that also goes along with a gradual decrease in the lipase value as either destruction of pancreatic tissue or some other mechanism occurs which lowers the lipase. It usually stays up for only 10 to 14 days after the attack of acute pancreatitis.

He had an increased white count with fever. This could be an infectious process going on in the pancreas. He had a history of previous gallbladder disease, and that is often seen in patients who develop acute pancreatitis. He had slightly increased serum bilirubin. He had shaking and convulsions which developed terminally. This could be explained by the low calcium. He had epigastric tenderness, and he had a terminal diarrhea which is often seen in cases of acute pancreatitis that terminate fatally. The last two or three days are often characterized by diarrhea.

The other thing which concerned me in this protocol was the increasing azotemia. Certainly, with the disorientation that the man had, I think we can say that he had uremia. Acute pancreatitis can produce lysis of viscera inside the abdominal cavity with areas of either necrosis or gangrene and associated with elevation of the NPN. So I think that I'll hang my hat on acute pancreatitis with secondary uremia and death in this patient due to uremia.

Dr. Delp: Thank you, Smith.

CLINICAL DISCUSSION

Dr. C. Frederick Kittle, assistant professor of surgery: All the interesting things that were pointed out are in retrospect, with regard specifically to renal function, the specific gravity at the time of admission in a patient who was febrile, who was dehydrated, and whose urinalysis also showed a heavy trace of albumin. I think it would indicate that he did have a considerable degree of renal insufficiency at that time.

If we pursue the urinalysis a little further, we notice that there were always blood cells present, even in amounts up to 10 to 20 white blood cells per high power field. This is much more than should be present on the routine urinalysis. Particularly with such a large number of cells per high power field, one would have to say that the urine was infected.

Along with the renal aspects were the hypocalcemia and elevated phosphate. He was seen by Dr. Valk, who thought that he had an appreciable degree of renal insufficiency because of these facts that I have just enumerated, and who directed attention to increasing the serum calcium by continuing the administration of calcium gluconate.

If we turn aside from the renal aspects for a moment and consider the entire problem this man presented, we have a rather long case. For 9 or 10 days there's a patient who had a persistently febrile course with abdominal symptoms and abdominal findings on examination, characterized by nothing specific as far as any one diagnosis is concerned. Consideration was given to the possibility of pneumonia when he first entered because of the appearance of some rales in his chest. The site of these was noted on the x-ray examination. As you are aware, an ileus can occur with severe pneumonia, and this is one of the initial suggestions made in this particular case. His subsequent course could not bear this out.

The next possibility that suggested itself by the prolonged distention was that of intestinal obstruction either of the small intestine or of the large intestine. He was sigmoidoscoped immediately on admission with negative results. The fact that he had bowel movements and passed gas toward the end of his hospital stay would be evidence, I think, that any obstruction was not a complete one.

So I think we should turn from the diagnosis of an obstructive lesion to some sort of inflammatory lesion within the abdomen. The diagnosis of pancreatitis is the diagnosis that answers all of these criteria as well as fitting in with the various laboratory results that were obtained. The lowered calcium in cases of acute pancreatitis is something that perhaps is ignored too frequently.

Actually, what happens is that with the release of the trypsinogen that becomes activated into trypsin and the subsequent digestion of the pancreas with the break through of the pancreatic enzymes into the abdominal cavity, lipase is released. Lipase then proceeds to break down the fat, the omentum, and the mesentery with the subsequent formation of a large number of unsaturated fatty acids. These unsaturated fatty acids react with the calcium and the serum with the formation of soap in the mesentery and in the omentum and also a marked hypocalcemia. This patient first began having irregular twitching episodes on the third day before death and continued to more generalized convulsions on the day of his demise.

Regarding indications for operation in this particular patient, I think that perhaps some difficulty was encountered because the patient had been sick for almost a week before he was admitted to this hospital. If he had been observed for several days, the fact that his course was becoming worse would in itself have been indication for a laparotomy. At the time of laparotomy, the diagnosis could have been established and therapy indicated. I think it would be better not to proceed with the discussion of therapy in this particular case until after the pathology report.

Dr. Delp: Dr. Allen, would you care to make any comments concerning this patient before we see the pathology report?

Dr. Max Allen, associate professor of medicine: My thought after seeing him was the possibility of pneumonia as the reason for the ileus. I think usually in the patient who has disease above the diaphragm, pneumonia is responsible for ileus. That disease would be fairly obvious. Other causes would include both renal colic and gallstone colic, back injuries, and medications.

I think the tendency for hypocalcemia and convulsions might be aggravated in this patient by uremia and the slight alkalosis which he had originally. This later probably was followed by the acidosis of uremia.

When I first saw him, I talked to one of the relatives, who led me to believe that this man was a stoic individual in that he would tend to minimize his pain or discomfort. I charted this because I thought his general appearance might be one of that sort. Finally, I think that, as someone mentioned, this man very easily could have previously been hypertensive so that any degree of shock which might occur in his early illness would certainly be a factor to be considered in precipitating acute tubular necrosis.

Dr. Delp: The total length of time between the onset of illness and death was about 14 days. Would anyone else like to comment before Dr. Firminger tells us about this case?

Dr. Stanley Friesen, associate professor of surgery and oncology: Although I think the likely diagnoses have been mentioned, no one has mentioned acute appendicitis which, in my opinion in this particular patient, could be a very likely finding—appendix rupture followed by peritonitis.

Dr. Delp: The third year medical student wrote the following diagnostic possibilities on the chart: (1) acute pancreatitis, (2) mechanical ileus due to small bowel obstruction, (3) rule out ruptured appendix with retroperitoneal appendiceal abscess and peritonitis, volvulus, (4) chemical peritonitis, (5) acute diverticulitis, and (6) acute nephritis.

PATHOLOGY REPORT

Dr. Bernard L. Klionsky, resident in pathology: In the abdomen there were extensive areas of fat necrosis throughout the peritoneum, omentum, mesentery, and surface of the diaphragm and extensive swelling, necrosis, and hemorrhage in the region of the pancreas, which was almost completely destroyed.

There was, in addition, a chronic cholecystitis; a small, shrunken, thick-walled gallbladder, which contained one small mulberry-type stone. An additional stone was in that segment of the common bile duct which was shared in common with the

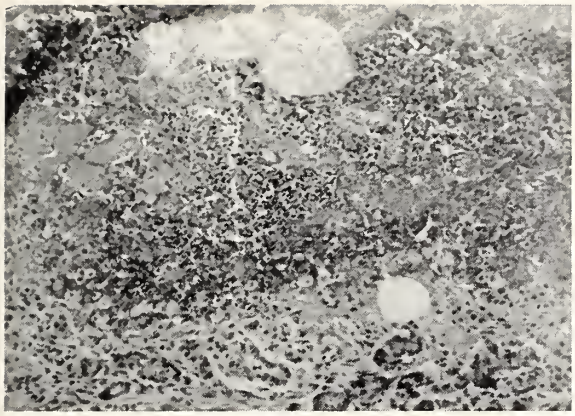


Figure 3. Photomicrograph of pancreas section, showing acute hemorrhagic pancreatitis.

main pancreatic duct. In other words, these two had, for about one centimeter, a common opening to the ampulla of Vater, and there was a small stone within that common channel.

The other main finding was in the kidneys, which were extremely large and pale, weighing over 500 grams. There were a number of small cysts grossly. The diagnosis we made was an acute nephrosis of some kind. Microscopically, we will see exactly what it was.

The heart was enlarged, weighing 450 grams. The other findings, I think, are relatively incidental. There was an ascaris worm in the intestines. In regard to intestinal obstruction, an adhesive band was between the cecum at a point 15 cm. distal to the ileocecal valve. A band was between that point and the edge of the liver, which band had produced a dilatation and a partial obstruction of the proximal cecum. There also were a few diverticuli but no evidence of diverticulitis. Another finding I neglected to mention was a massive partial infarct of the liver, and there were many thrombosed intrahepatic branches of the portal vein. We saw no other gross thrombi.

Dr. Harlan I. Firminger, associate professor of pathology and oncology: Though we looked carefully for thrombi in the pancreatic veins and were unable to see them grossly, microscopically they were apparent. There were organizing thrombi in veins in the pancreas due to inflammatory involvement of the wall of the pancreatic veins. Some of these had given rise to emboli which went to the liver and went into the portal vein in the liver and gave the picture of an organizing embolus in the wall of the portal vein. He did not have bacterial peritonitis with spread up to the lymphatics into the liver.

The kidneys, as Dr. Klionsky mentioned, were large and pale, typical of acute nephrosis. Microscopically, we found evidences of a healing lower nephron hypoxic type of nephrosis with regenerating tubules. Actually, there were a fair number of mitotic figures seen. One area showed a granuloma-like formation which actually was cast material, perhaps of renal epithelial cells which had died and had been sloughed but also albumin and sometimes pigmented material which obstructed the tubule, causing its wall to break down and be extruded into the adjacent tissue. This takes time, and it helped us to date the time at which the actual nephrosis came about.

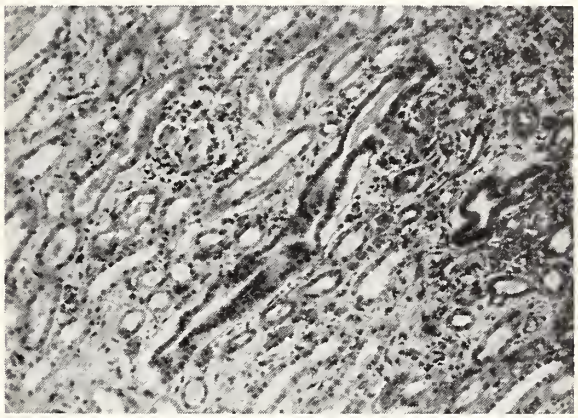


Figure 4. Photomicrograph of kidney section, showing healing lower nephron nephrosis with regenerating tubules and cast material.

Another lesion which one finds in this type of nephrosis is a chronic inflammatory infiltration at the corticomedullary junction about the veins and venules. Here one sees a mass of chronic inflammatory cells projecting into a venule. This is also typical and helped us to date the onset of this lesion.

If I may reconstruct the case as I see it at the moment, it is essentially this. This is a 64-year-old man who had probably liked to eat most of his life. He had some metabolic defect which many people have of putting on fat, and he became obese. Along with this, probably he developed some stones in his gallbladder. Indeed, he had difficulty some three years before. A gallstone of similar type to that found at autopsy produced some symptoms and jaundice and apparently passed without further difficulty.

After eating Thanksgiving Day dinner, the gallbladder was vigorously stimulated by the meal, and another stone was extruded into the common duct but was unable to get through the ampulla. The patient was unfortunate enough to have the anatomic relationship many people have, of the pancreatic duct joining with the common duct about one centimeter before it entered the ampulla and into the duodenum. The stone lodged at this point and produced obstruction. The obstruction produced a reflux of bile perhaps, but at least it increased tension, this produced back pressure in the pancreas, and

there was leakage of pancreatic enzymes out into the surrounding tissue, and an acute hemorrhagic pancreatitis developed.

He must have had severe pain at that time. He must have been a stoic individual. Some older persons do not show much pain, possibly due to elevated thresholds.

In any event, I suspect he may well have gone into shock at that time. Shock for him might have been present with a fair blood pressure. Subsequently he was admitted to the hospital and treated. Almost surely he had oliguria at that time. On arrival here there was evidence of uremia, but the oliguria was beginning to clear. I'd date the acute renal damage 10 to 14 days before death, based on the morphologic changes we see. I think he could have had shock and a hypoxic type of change at the time of the initial onset of disease and that that alone—the prolonged shock for six or so hours, followed by recovery—will produce a lower nephron nephrosis such as we see here and could well account for the renal difficulty.

I think practically all of the laboratory findings which were described can be explained on the basis of the morphologic changes we see. The elevated white count and the renal disease are understandable. I think the phosphorus retention could be explained by the renal damage, and that in itself would tend to lower the calcium, although I think the lowered calcium is also part of the disease in which calcium is taken up with fatty acids. There was some disturbance in liver function. I don't know whether or not that's explained on the basis of the peritonitis.

Dr. Delp: Any questions of Dr. Firminger?

Q: Were any vascular changes noted in the kidneys?

Dr. Firminger: Not particularly.

Dr. Delp: I'd like to have a few comments about the treatment of acute pancreatitis, Dr. Kittle.

Dr. Kittle: I think treatment should be directed in two areas: first the local treatment and then the general treatment. The local treatment should be aimed at those measures which will decrease pancreatic secretion. In this particular patient, that was managed by gastric suction, aspirating the gastric contents so that they did not stimulate gastric secretion. One should also give frequent doses of atropine. The other local measures are those designed to control the ill effects of the enzymes after they have spilled out in the peritoneal cavity. Generally there is peritonitis, so massive doses of antibiotics should be given.

With regard to the general supportive treatment, these patients usually have a reduced blood volume. They go into shock because of the exudation of large amounts of fluid from the mesentery and the peritoneal surfaces. This fluid must be replaced by blood or by plasma. I think that probably this patient may have had a reduced blood volume, even though his blood counts were within normal limits. I think there is every indication that his blood volume was decreased. Supportive therapy has to be considered to maintain normal blood volume. If hypocalcemia develops, repeated injections of calcium gluconate are necessary.

If the diagnosis can be made short of a laparotomy, then it is usually thought that conservative management should be tried first.

If the patient continues to become worse and does not improve on conservative measures, then laparotomy is usually indicated, at which time some sort of surgical drainage of the extrahepatic biliary tract should be attempted. If there are any localized abscesses or localized collections of fluid, for example in the lesser omental cavity or in the peritoneal cavity elsewhere, those are drained. There is no really satisfactory treatment other than drainage of the extrahepatic biliary tract and any abscesses.

A serious implication of acute pancreatitis is recurrence of the attacks and possible development of chronic pancreatitis.

ANATOMICAL DIAGNOSIS

Primary

Chronic cholecystitis and cholelithiasis, with two small mulberry stones, one at the ampulla of Vater beyond the confluence of the common bile duct with the duct of Wirsung.

Dilatation of the common bile duct, slight.

Acute hemorrhagic pancreatitis, advanced, with fat necrosis involving the mesentery, omentum, parietal peritoneum of the diaphragm, hilus of the left kidney, and periadrenal fat on the left (history of onset of abdominal discomfort and distention 14 days before death).

Organizing thrombotic occlusion of pancreatic vein.

Organizing emboli occluding intrahepatic branches of the portal vein.

Recent massive incomplete infarct involving both lobes of the liver.

Acute passive congestion of lungs, slight, and of liver, moderate.

Acute hypoxic nephrosis (history of uremia and severe electrolyte imbalance for five days before death).

Focal submucosal hemorrhages of the rectum.

Fibrous adhesions between the midportion of the ascending colon and the liver with retrograde dilatation of the colon.

Atelectasis of the posterior basal portions of all lobes of the lungs.

Diffuse emphysema of remaining portions of the lungs.

Acute hyperplasia of the spleen, slight.

Focal depletion of lipoid in the cortex of the adrenal glands.

Acute ulcer of the stomach and acute ulcerative esophagitis (history of repeated emeses beginning 14 days before death and of intubation for 8 days).

Acute hemorrhagic cystitis and urethritis (history of indwelling catheter for 5 days before death).

Recent healing linear incisions in the left femoral regions and above both medial malleoli, multiple puncture wounds on forearms and dorsum of hands (history of intravenous injections for 8 days before death).

Segment of Miller-Abbott tube in ileum.

Accessory

Obesity, 225 pounds.

Arteriosclerosis of the cerebral arteries, moderate; and of the aorta and coronary arteries, advanced, with marked narrowing of anterior descending branch of the left coronary artery.

Sclerosis of the anterior leaflet of the mitral valve, moderate.

Recent and organizing hemorrhages beneath an arteriosclerotic plaque in the circumflex branch of the left coronary artery.

Hypertrophy and dilatation of the heart, 450 grams.

Foci of fibrosis in myocardium.

Fat infiltration of the myocardium, slight.

Accessory ostium of the right coronary artery.

Calcified subpleural nodule in the upper lobe of the right lung and caseocalcified tracheobronchial lymph node, right.

Small fibrous nodules in the parenchyma of the spleen.

Ascariasis of the intestine, 1 adult worm.

Diverticulosis of the sigmoid colon, slight.

Nodular hyperplasia of the prostate with median bar formation, slight.

Multiple retention cysts of both kidneys.

Accessory renal artery, slight.

Osteoarthritis of the spine, slight.

Osteoporosis of ribs, vertebrae, and sternum, slight.

Hallux valgus with prominence of the head of the first metatarsal of the left great toe.

Flexion deformity of the proximal interphalangeal joint of the right fifth finger.

Deformity of the bridge of the nose (history of broken nose).

BIBLIOGRAPHY

Opie, E. L.: Bull. Johns Hopkins Hosp. 12:182, (April, May, June) 1901.

Is each physician conscious of the fact that good public relations for the medical profession begins in *his* office?

McIlvain Career Investigatorship

The Board of Directors of Community Studies, Inc., and the Department of Medicine of the University of Kansas School of Medicine announce the establishment of the McIlvain Career Investigatorship at the University of Kansas School of Medicine and the appointment of Dr. Santiago Grisolia as the McIlvain Career Investigator. The appointment became effective May 1.

A grant of $54,000 has been made to the school by Community Studies, Inc., from a trust established by the late Mrs. Frederic E. McIlvain in 1950. Dr. Sam Roberts, medical advisor to the trustees of the estate, is assisted by a committee composed of Doctors E. Grey Dimond, Kurt Reissmann, Stanley Morest, Blaine Hibbard, Harold Grady, and Robert E. Bolinger.

Dr. Grisolia was selected as the investigator on the basis of his research record and because his specific field—enzyme, hormone, and electrolyte changes in heart disease—seems to hold promise in the study of heart disease. He is a graduate of the University of Barcelona Faculty of Medicine and is coming to Kansas from the Enzyme Institute, University of Wisconsin, where he has been assistant professor of physiological chemistry. At the Kansas school he will be an associate professor of medicine and biochemistry and a member of the Department of Medicine and of the Cardiovascular Laboratory.

Successful Cancer Conference

The Sixth Annual Mid-West Cancer Conference, held April 1 and 2 at the Broadview Hotel, Wichita, was characterized by a record breaking attendance and a good, practical scientific program. Although most of those attending were from Kansas, physicians from neighboring states were also registered.

Dr. Karl E. Voldeng, Wellington, president of the American Cancer Society, Kansas Division, announced that the 1955 conference has been set for March 24 and 25. That session also will be held at the Broadview Hotel, Wichita. Dr. Homer L. Hiebert, Topeka, has been named as chairman of the committee to arrange the scientific program.

Only 100 years have passed since Kansas appeared on the map. It was in 1854 that the President of the United States, Franklin Pierce, signed an act establishing the Territory of Kansas. It was in 1859 that the governor of the Territory of Kansas approved the Act of Incorporation of the Kansas Medical Society.

Patronize JOURNAL advertisers.

ACTIVITIES OF MEMBERS

Dr. Charles F. Haughey, who has been taking postgraduate work in surgery in Chicago and New Orleans, returned recently to his practice in Tribune. Dr. R. N. Todd, who cared for Dr. Haughey's practice, will go to Kansas City in June to begin a residency in radiology at the University of Kansas Medical Center.

Dr. H. M. Wiley, Garden City, spoke on cancer before the Dighton Rotary Club last month and also addressed an all-county cancer meeting there the same evening.

Dr. R. E. Riederer, Olathe, addressed the Central Parent-Teacher Association there recently on "A Medical Man Looks at Health and Accident Insurance."

Dr. George M. Gray, Kansas City, observed his 98th birthday anniversary in March. A story on the activities of the retired physician was carried in the *Kansas City Kansan*.

Dr. Edward Goldsich, who has practiced in St. Marys for four years, has announced plans to move to Kansas City, Missouri, for a residency at St. Luke's Hospital. Dr. John Montague, a graduate of St. Louis University School of Medicine who interned 18 months at Mercy Hospital, Denver, will take over Dr. Goldsich's practice.

Dr. Warren F. Bernstorf and Dr. Lynn C. Hartman took part in the Cowley County Health Education Workshop held at Winfield, March 18 and 19.

Dr. Eugene C. Kane, who has been associated with the Scott City Clinic during the past two years, has announced his resignation from the staff of the clinic, effective June 30.

Dr. David Robinson, of the University of Kansas Medical Center, presented a paper, "Prevention and Treatment of Scars," at a meeting of the Missouri State Medical Association held last month.

Dr. George W. Hammel, El Dorado, addressed the nurses' club in that city recently on the subject of "Cardiac Emergencies" and showed a film, "Wonder Engine of the Body."

Dr. Herbert R. Schmidt, Newton, returned recently from a trip to Paraguay where he visited his brother, Dr. J. R. Schmidt, and Mrs. Schmidt who are part of a Mennonite medical team working among lepers there.

Dr. C. V. Black, Pratt, addressed the grade school Parent-Teacher Association there recently on the subject of public health.

Dr. Raymond W. Lance, who practiced in Moline before entering service with the Air Force, was recently released from active duty and is opening an office in Arma.

The Lions Club in Girard recently heard a talk on "Community Health" given by Dr. Robert W. Friggeri.

Dr. Dick B. McKee, representing the Kansas State Board of Health, was one of the speakers at a youth health education conference held at Kansas State Teachers' College, Pittsburg, last month.

Dr. W. Clarke Wescoe, dean of the University of Kansas School of Medicine, was principal speaker at a health education tea given by the Woman's Auxiliary to the Wyandotte County Medical Society in Kansas City recently.

Dr. John L. Lattimore has been named to serve as chairman of the Legislative Committee of the Topeka Chamber of Commerce.

Dr. Richard L. Sutton, Jr., of the University of Kansas Medical Center, spoke on "Early Stages, Recognition, and Treatment of Cancer of the Skin" before the Alabama Division of the American Cancer Society at a meeting held recently in Birmingham.

At an area meeting of the American Cancer Society held in Salina recently, Dr. John H. Lathrop, Concordia, was elected medical representative from the 10th district to the state board. Dr. Donald D. Dieter,

Salina, presented a film at the meeting, "Cancer—A Research Story," and conducted a question and answer session.

Dr. Lucien R. Pyle, Topeka, took part in a panel discussion on interprofessional relations during the annual meeting of the Kansas Pharmaceutical Association held in Topeka last month.

Dr. Karl E. Voldeng, Wellington, was speaker at a recent meeting of the Sumner County Medical Assistants' Society.

Dr. Frederick P. Wolff, who is in the service and is stationed at Camp Pendleton, California, was given a leave of absence in February to return to Pratt to aid Dr. M. E. Christmann who is recovering from injuries received in an automobile accident.

Dr. John P. White and Dr. Earl A. Martin, Parsons, were speakers at a three-day conference of nurses of the Southeast Kansas area held in Parsons in February.

Dr. Thomas L. Foster, Halstead, spoke on "Mental Hygiene" before a meeting of the Parent-Teacher Association in Canton in February.

Dr. DeWitt S. Lowe, Hiawatha, former medical missionary for the Presbyterian Church in South Korea, was the principal speaker at the Topeka observance of World Day of Prayer in March.

Dr. James A. Dugger, Topeka, recently became a diplomate of the American Board of Pediatrics.

Dr. Janet L. Holloway has returned to practice in Wichita after having completed a two-year residency in anesthesiology at the Lahey Clinic.

Dr. and Mrs. John L. Lattimore, Topeka, recently returned from a six weeks tour of Europe.

Dr. Robert M. Knox, who practiced in Wamego before going to Iowa Methodist Hospital in Des Moines for a residency in surgery, has announced plans to join the staff of the Nelson Clinic, Manhattan.

Dr. Joseph Gendel, Topeka Medical Center, has completed the examinations of the American Board of Orthopedics and is now a diplomate of the Board.

Dr. James G. Hughbanks, Dr. Albert E. Bair, and Dr. Vernon M. Lockard took part in a medical forum on the subject of cancer in Independence in March. A series of panel discussions on medical topics is being sponsored there by the Independence Rotary Club.

Dr. and Mrs. C. Omer West, Kansas City, left in February for a flight around the world.

Dr. N. C. Nash, Wichita, spoke on "Difficulties Encountered in Upper Gastrointestinal Tract Examinations" before a spring meeting of the Jackson County (Oklahoma) Medical Society in Altus.

Dr. Simon E. Hershorn, who has been serving in the Army, has received his discharge and has returned to practice in Wichita.

Dr. Lottie F. Law, Hill City, who has been practicing 52 years, announced her retirement recently. Two years ago, on the occasion of her golden anniversary in practice, 2,000 members of the community joined in a celebration in her honor. Her son, Dr. Findley Law, practices in Bird City.

Dr. John B. Dixon, Parsons, presented a paper on "Headaches" before a meeting of the Southeast Kansas Optometric Society on March 29.

Dr. J. D. Smith, who has been practicing in Hugoton during the past year, announced a change of location on April 1. He is now practicing in Satanta.

Dr. Edward A. Marrs, who has practiced in Sedan for 32 years, has retired and has moved to Colorado Springs.

Dr. Mervin T. Sudler and Dr. Monti L. Belot, Lawrence, announce that Dr. Glen A. Lessenden,

formerly of Tonganoxie, is now associated with them in practice.

Dr. E. Grey Dimond, of the University of Kansas Medical Center, is the author of a book, *Electrocardiography,* published last month by the C. V. Mosby Company, St. Louis.

Dr. Paul Lorhan, of the University of Kansas Medical Center, was speaker at a meeting of the Lincoln County, Nebraska, Medical Society recently. His subject was "Obstetrical Anesthesia."

Dr. Clarence H. Benage, Pittsburg, has been named by the Board of Trustees of the American Medical Association to the Commission on Improvement of Patient Care. The commission is made up of representatives of the A.M.A., the American Hospital Association, the American Nurses' Association, and the National League for Nursing.

Dr. Robert A. Jordan, Lawrence, has been appointed to the medical staff of the University of Kansas School of Medicine as assistant professor of medicine, director of the medical service of the outpatient department, and director of the student health center.

BLUE SHIELD

New Educational Program

Beginning in April, Blue Cross and Blue Shield Plans across the nation heralded the start of their first national program of public education.

A program of national magazine space has been outlined in three publications that will enable both Blue Cross and Blue Shield to explain their unique qualities in the field of prepayment health care.

Blue Cross began its advertising program with two-color, two-page spreads in LIFE, SATURDAY EVENING POST, and LOOK. Blue Shield is using the same media in different issues of the same magazines. Although Blue Cross and Blue Shield will have individual campaigns, there will be cross references in each advertisement to each other.

"The need for bringing the Blue Cross and Blue Shield stories to the American public in some continuing, co-ordinated way has become increasingly evident," Frank E. Smith, director of the Blue Shield Commission, points out. "Blue Cross and Blue Shield," he said, "were organized primarily as community services and not as commercial ventures. They are dedicated to serving the American people and fulfilling a social purpose. The new program of national education will attempt to point this out."

It is believed by the plans endorsing the national program that a strong conviction can be built in readers' minds to help them properly evaluate the welter of promises being made today in the hospital and medical protection market place.

Space in the national publications gives Blue Cross and Blue Shield the latitude necessary for their stories, as well as affording the plans the most effective and practical way to educate their present members and prospective members.

"Logically, if members really understand what their membership means, they will make a greater effort to retain it," Smith said. "At a cost far less than that offered by any other method, the new means of education will contact these members regularly in their homes. Wives, as well as husbands, will be exposed to the meaning of belonging to Blue Cross and Blue Shield."

Present plans call for nine Blue Cross messages in LIFE, eight in the POST, and eight in LOOK. The Blue Shield messages will appear five times in LIFE, five times in the POST, and four times in LOOK. Combined circulation of the three magazines for any one issue is 71 million.

Contributions from local Blue Cross and Blue Shield Plans are financing the national advertising program. Kansas Blue Cross-Blue Shield has not committed itself to participate at this time.

Dr. Howard Schriver, president of the Blue Shield Commission, has this to say about the national educational program: "We have felt for a long time that one barrier to even greater growth has been our lack of communication with the millions of people who want to know about Blue Shield yet are not exposed to the organization's story. When the first national Blue Shield advertisement appeared, we took the first major step toward telling our story on a national multimillion level."

COUNTY SOCIETIES

A meeting of the Tri-County Society was held at Harper in February. The program featured case reports by physicians from Sumner, Harper, and Barber counties.

Dr. Alfred Tocker, Wichita, was guest speaker at a recent meeting of the Barton County Society held at the Great Bend Country Club. His subject was

"Surgery of the Chest and Heart." Members of the Pawnee County Society were guests at the meeting.

Dr. David Culp, of the State University of Iowa College of Medicine, spoke on "Treatment of Carcinoma of the Prostate," before the Sedgwick County Society at its March meeting at the Lassen Hotel, Wichita. In April the group heard Dr. F. H. Ellis, of the Mayo Clinic, who discussed "Breast Surgery."

Dr. James J. Marchbanks, Oakley, was named president of the Northwest Kansas Society at a meeting held in Colby recently, and Dr. Edward E. Johnson, Norton, was chosen as vice-president. Dr. John S. Oney, Oakley, is secretary of the group.

Dr. W. J. Singleton, LaCrosse, was elected president of the Rush-Ness Society at a meeting held recently at the Rush County Memorial Hospital. Dr. J. H. Baker was named secretary, and Dr. F. K. Bowser will serve as delegate to the state meeting with Dr. Kenneth McLain as his alternate.

A combined meeting of the medical societies and the auxiliaries of the First District was held at Centralia in February. Guest speakers were Dr. Lucien R. Pyle, Topeka, president of the Kansas Medical Society, and Mrs. I. Joseph Waxse, Oswego, president of the auxiliary for the state. The meeting was arranged by Dr. W. L. Anderson, Atchison, and Mrs. C. M. Barnes, Seneca, councilors for the two groups.

"The Clinical Course of Poliomyelitis" was the subject of a paper given the Shawnee County Society by Dr. J. G. McQueen, Iowa City, at a meeting held in Topeka on March 1. At a meeting held on April 5 Dr. John Berry, Denver, spoke on "Cirrhosis of the Liver." Dr. Howard U. Kennedy was nominated as a trustee for Blue Cross-Blue Shield.

The Shawnee County Society has completed organization plans for civil defense. Members are assigned to one of three hospital units. Unit captains are Dr. Byron J. Ashley, Dr. D. R. Bedford, and Dr. George Hopkins. Assistant captains are Dr. Floyd C. Taggart, Dr. Charles E. Joss, and Dr. William Delaney. Dr. Dwight Lawson and Dr. Lucien R. Pyle are captain and assistant captain of a mobile support unit.

Five first aid teams have been assigned to different areas. Captains are Dr. Vernon M. Wiksten, Dr. A. J. Revell, Dr. L. L. Robbins, Dr. Clark Case, and Dr. Shirley Clark, and assistant captains are Dr. C. K. Schaffer, Dr. Evalyn Gendel, Dr. Seth Hammel, Dr. John Adams, and Dr. Lloyd Schwartz.

Members of the Harvey County Society were guests of the new Prairie View Hospital, Newton, at a dinner meeting held last month at the hospital. Additional guests were Dr. Thomas Morrow and Dr. F. Carter Newsom, Wichita, part-time staff psychiatrists at the hospital, and several Reno County physicians. The hospital, one of three mental institutions in this country operated by those of the Mennonite faith, contains 40 beds and will be open to persons of all creeds.

Dr. F. H. Ellis, of the Mayo Clinic, was guest speaker at a meeting of the Sedgwick County Society held at the Lassen Hotel, Wichita, on April 6. At the May meeting, on the 11th, members of the society presented a clinicopathological conference.

Members of the Johnson County Society discussed civil defense at a meeting held at the Milburn Country Club late in February. Dr. J. E. Schiffmacher, radiologist at St. Joseph Hospital, Kansas City, Missouri, spoke and presented a film depicting civil defense organization in Syracuse, New York. Dr. R. E. Riederer and Dr. G. J. Pierron were re-elected as president and secretary of the society.

A meeting of the Cowley County Society was held at the Arkansas City Country Club on March 18. Dr. Fred V. Hein, consultant of the Bureau of Health Education of the American Medical Association, spoke on "Physician Participation in the School Health Program." Members of the auxiliary were guests of the doctors at a dinner preceding the meeting.

A dinner meeting of the Nemaha County Society was held at Sabetha on April 8 with the two state legislators of that area as guests. Dr. Lucien R. Pyle, Topeka, led the discussion on legislative matters.

There are 220,104 physicians in the United States today, 156,433 of whom are in active practice, according to the A.M.A. On the basis of an estimated population of 160,000,000 in 1953, there is one physician actually engaged in the practice of medicine for every 1,000 persons.

THE MONTH IN WASHINGTON

Editor's Note. The following summary of Washington news was prepared by the Washington office of the A.M.A. for distribution to state and regional medical journals.

These spring days are growing into weeks that really count in Congress. Unless a bill deals with an emergency, it had better be well on its way through committees by now or its chances of enactment will fade rapidly as summer approaches.

For good or evil, a large amount of health legislation is well advanced, and if Congress holds to an average pace several bills affecting the medical profession are likely to become law in the next month or so. Here is the situation in brief:

MEDICAL DEDUCTIONS. Legislation to increase the amount deducted from taxable income for medical expenses is a part of the omnibus tax revision bill which cleared the House early and by a wide margin, but ran into some delay on the Senate side. This bill, with the medical deduction liberalization intact, should reach the White House in plenty of time.

HILL-BURTON EXPANSION. A move to make important changes in this bill developed in the Senate Labor and Welfare Committee, after the House had passed its version with some amendments. American Hospital Association proposed that the rather complicated House legislation be scrapped, and instead that the Hill-Burton Act be amended to (a) include rehabilitation centers and nursing homes, and (b) place a high priority on hospitals for the chronically ill. The A.H.A. idea immediately attracted support in and out of the committee. The new approach meant inevitable, but probably not fatal, delays.

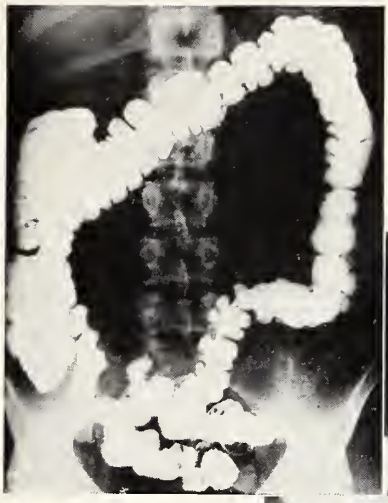
REINSURANCE. This proposal, once hailed as the keystone of the Eisenhower administration's health program, continued to encounter opposition. At one stage, of all the national associations to testify on reinsurance only American Hospital Association was giving it unqualified support. American Medical Association, the U. S. Chamber of Commerce, and national spokesmen for the insurance industry took about the same position: (1) Reinsurance alone cannot make uninsurable risks insurable. (2) The threat of federal control of medicine is inherent in any program that would bring the federal government in such close contact with medical practice. Dr. David B. Allman, representing the A.M.A. at the House hearings, emphasized that the association would welcome and co-operate in any movement carrying real promise of promoting voluntary health insurance.

HEALTH GRANTS. This is an administration plan to do away with the present categorical grants for identified projects, such as venereal disease control, and to substitute funds earmarked for three general purposes: (a) to maintain present programs, (b) to initiate new programs or to expand existing ones, and (c) to finance public or private experimental or pilot programs of national or regional significance. In both committees the question was whether to group the first and second type grants together, with the state health authorities deciding how to divide up the federal money among old and new projects. Funds for the third type grant—experimental—would be completely controlled by the surgeon general. One suggestion is to require approval of the state health officer for any experimental (type three) grant in his state. Another is to eliminate the third type grants altogether, letting the National Institutes of Health handle public health as well as other medical research grants.

SOCIAL SECURITY. American Medical Association, American Dental Association and a number of other national groups are fighting vigorously to prevent compulsory extension of Old Age and Survivors Insurance to physicians, dentists, and most other self-employed. Instead, they want the privilege of deferring income tax payments on that part of earnings placed in restricted annuities—the Jenkins-Keogh plan. A.M.A. also feels that there is no need for the bill's provision that pension rights be frozen during periods when the worker has been medically determined to be disabled. A better suggestion, the Association maintains, is to base pension rates on the 10 best working years, thus virtually eliminating the need for the controversial medical examinations. Prospects are good that social security will be extended, either with or without these changes.

VOCATIONAL REHABILITATION. Generally, Senate witnesses favor the administration's proposal to expand the federal-state programs, providing U.S. grants aren't cut. However, with no House bill introduced as of this writing, there is some doubt that, even if the Senate clears the measure, the House can find time to deal with it.

DOCTOR DRAFT AMENDMENT. This bill, an outgrowth of the Peress case, swept through the Senate without objection. It may be law by the time this is published. It would amend the Doctor Draft act to permit the services to keep on duty as an enlisted man, assigned to professional tasks, anyone called under the Doctor Draft act whose loyalty is questioned. Defense Department has promised to investigate such cases immediately, so that the man can be cleared promptly and offered a commission or discharged. The discharge would state that action was taken on loyalty grounds.



Normal Colon



Ulcerative Colitis



Atonic Colon

Smoothage and Bulk in Correcting Constipation

To initiate the normal defecation reflex, the "smoothage" and bulk of Metamucil® provide the needed gentle rectal distention.

Once the habit of constipation has been established, due to any of a large number of causes, it becomes a major problem. Self-medication with irritant or chemical laxatives, or repeated enemas, usually causes a decreased, sluggish defecation reflex and may result in its complete loss.

Rectal distention is a vital factor in initiating the normal defecation reflex, and sufficient bulk is thus of obvious importance in restoring this reflex. Metamucil provides this bulk in the form of a smooth, nonirritating, soft, hydrophilic colloid which gently distends the rectum and initiates the desire to evacuate. Metamucil demands extra fluid, imparting even greater smoothage to the intestinal contents.

It is indicated in chronic constipation of various types—including distal colon stasis of the "irritable colon" syndrome, the atonic colon following abdominal operations, repressions of defecation after anorectal surgery and in special conditions such as the management of a permanent ileostomy. Metamucil is the highly refined mucilloid of Plantago ovata (50%), a seed of the psyllium group, combined with dextrose (50%) as a dispersing agent.

The average adult dose is one rounded teaspoonful of Metamucil powder in a glass of cool water, milk or fruit juice, followed by an additional glass of fluid if indicated.

Metamucil is supplied in containers of 4, 8 and 16 ounces. It is accepted by the Council on Pharmacy and Chemistry of the American Medical Association. G. D. Searle & Co., Research in the Service of Medicine.

Treatment of Hepatic Insufficiency

Helen Jones, M.D.

Tucson, Arizona

"He who would know correctly before hand those that will recover and those that will die, and in what cases the disease will be protracted for many days and in what cases for a shorter time, must be able to form a judgment from having made himself acquainted with all the symptoms and estimating their powers in comparison with one another." Hippocrates' *Book of Prognostics.*

A consideration of the immediate effects of injury upon composite organs reveals that, in general, those cells which are most highly differentiated, and which endow the organ with its characteristic function, are also those which are most susceptible to damage. Nowhere is this more evident than in the liver.[1]

The term "hepatic insufficiency" may be defined according to the usage of the individual observer of hepatic disease. There would probably be a uniform acceptance of the term for the comatose or disoriented patient with a severe hepatic disorder, such as diffuse necrosis or cirrhosis. It might also be used to apply to more serious clinical states associated with diffuse parenchymal hepatic disease, with jaundice and ascites, but without obvious mental depression or aberration. It would seem helpful to define "hepatic insufficiency" as that state in which the patient's normal economy and activity is seriously interfered with because of a diffuse hepatic injury; in other words, a state in which the functions of the liver are unable to meet the general needs of the individual.[2]

Cirrhosis of the liver with ascites and massive hemorrhage presents a well known clinical picture, but the less severe forms of chronic liver disease are usually not diagnosed. Yet it is probable that various types of low-grade chronic liver disease are quite common. Our failure to recognize these cases clinically is apparently due to an absence of a clearly defined and well established clinical picture. Symptoms and physical findings are not striking, and definitive laboratory procedures are often lacking. However, it does not seem reasonable to assume that disease of an organ which plays such a varied and important role in the body economy as the liver is entirely "silent" and without clinical manifestations.[3]

Consideration of the clinical types of liver disease fully supports the conclusion derived from a study of its pathology, that the same type of liver injury may, on occasion, be caused by several different factors. The clinical manifestations of liver injury from widely different causes may be indistinguishable, and it is only by attention to associated general effects of the causative agents or exposure to special conditions that the exciting cause of the illness can be recognized. Only in the broadest sense can the clinical manifestations of liver injury be related to the particular pathological lesion present.[1]

The earliest lesion in massive hepatic necrosis shows gross congestion of the portal tracts, periportal hemorrhage, and swollen dead parenchymal cells throughout the lobule. It seems that in this condition the parenchyma has swollen to such an extent as to arrest completely the intralobular circulation. Death of every parenchymal cell throughout the lobule—massive necrosis—is inevitable. A similar impeding of the intralobular circulation is produced by fatty infiltration. This is sufficient only to lead to quiet degeneration of the parenchyma.[4]

Necrosis of the liver is followed by fibrosis, of which there seem to be two distinct types. Post-necrotic scarring, which usually follows the massive type of necrosis, leaves an organ cut into by coarse bands of fibrous tissue with separate areas composed of normal liver lobules. The other is the diffuse fibrosis which begins as a thickening of the reticulin fibers in the region of the portal tract or central veins and involves every lobule. It is clear that the anatomical form of the original hepatic lesion has a bearing on the consequences of hepatic injury, and it would seem also to have an important influence on survival in patients with liver disease.[5]

From the evidence now available, it is believed that the prognosis in cases of so-called "alcoholic" cirrhosis is related to the degree of portal fibrosis, bile duct proliferation, and adenoma formation with resultant shrinking and distortion of normal liver parenchyma. These changes roughly indicate the extent of previous destruction of liver cells. In other words, the reversibility of hepatic insufficiency would appear to depend upon the remaining amount of liver parenchyma not damaged beyond repair. There is little doubt that a fatty cirrhosis, in which the enlargement is due mainly to fat rather than to adenomata and connective tissue, offers a much better prognosis than one of the latter type, even though it is still enlarged.[2]

This is one of 11 theses, written by fourth year students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group judged to be the best by the faculty at the school.

Thesis written while the author was a fourth year student. Dr. Jones is now interning at St. Mary's Hospital and Sanatorium, Tucson, Arizona.

Meats-in-a-Can

and Kitchen-Cooked Meats... Comparative Nutritive Values

From a practical dietary standpoint, meats-in-a-can—preserved by commercial canning—are nutritionally interchangeable with meats of like variety prepared in the home.[1] For taste appeal, for economy and "keeping" quality, and for household convenience, meats-in-a-can are advantageous in many respects.

As the comparative data here shown indicate, kitchen-prepared meats and similar meats-in-a-can are closely alike in the amounts of various nutrients they provide.

COMPARATIVE COMPOSITION OF KITCHEN-COOKED AND COMMERCIAL-CANNED MEATS
(Nutrient Amounts per 100 Grams)

	*Kitchen-Cooked Ham[2]	**Canned Ham[3] (Chopped, Cured)	Kitchen-Cooked Beef Round[2]	Canned Roast Beef[2]
Water	50%	50%	59%	60%
Protein	21 Gm.	20 Gm.	27 Gm.	25 Gm.
Fat (ether extract)	28 Gm.	20 Gm.	13 Gm.	13 Gm.
Niacin	4.0 mg.	4.3 mg.	5.5 mg.	4.2 mg.
Riboflavin	0.21 mg.	0.19 mg.	0.22 mg.	0.23 mg.
Thiamine	0.46 mg.	0.40 mg.	0.08 mg.	0.02 mg.

*Values after conversion from 42% to 50% water basis.
**Values after conversion from 58.69% to 50% water basis.

Experimental studies have shown that the processing which meats-in-a-can undergo leads to little if any greater vitamin losses than does home-cooking of similar cuts of meat. In general, meats-in-a-can retain of their original vitamin content approximately:

60 to 80 per cent of thiamine
90 to 100 per cent of riboflavin
90 to 100 per cent of niacin
80 per cent of biotin
70 to 80 per cent of pantothenic acid.[4,5]

During storage for customary periods, at usual warehouse temperatures, meats-in-a-can show little, if any, further vitamin loss except in thiamine. Even thiamine, a highly thermolabile vitamin, was 52 per cent retained in pork-in-a-can after ten months' storage at 80° F. Retention of the vitamin was notably greater when the canned pork was stored at 38° F.

Since meats-in-a-can are thoroughly cooked in processing, they may be consumed as purchased, merely warmed or mildly cooked. When the meat is moderately cooked in preparation for consumption, little or no further loss in vitamins need to occur.

Recent studies show that meats-in-a-can are excellent sources of needed amino acids.[6] The 18 amino acids determined in these studies appeared in similar ratio and amounts in canned beef, pork, and lamb as in the respective fresh or home-cooked meats.

1. Howe, P. E.: Foods of Animal Origin, Handbook of Nutrition, American Medical Association, ed. 2, Philadelphia, The Blakiston Company, 1951, p. 637.

2. Watt, B. K., and Merrill, A. L.: Agricultural Handbook No. 8, United States Department of Agriculture, 1950.

3. Schweigert, B. S.; Bennett, B. A.; Marquette, M.; Scheid, H. E., and McBride, B. H.: Food Res. *17*:56 (Jan.) 1952.

4. Rice, E. E., and Robinson, H. E.: Am. J. Pub. Health *34*:587 (June) 1944.

5. Schweigert, B. S.: Am. Meat Inst. Foundation, Circular No. 8, Nov. 1953.

6. Schweigert, B. S.; Bennett, B. A.; McBride, B. H., and Guthneck, B. T.: J. Am. Dietet. A. *28*:23 (Jan.) 1952.

The Seal of Acceptance denotes that the nutritional statements made in this advertisement are acceptable to the Council on Foods and Nutrition of the American Medical Association.



American Meat Institute
Main Office, Chicago . . . Members Throughout the United States

Symptomatology is the expression of deranged function and, although gross alterations in structure lead to such derangements, disturbances of function may occur from lesions too subtle to cause structural alterations. By analyzing the symptomatology of established pathological lesions, it is possible to see its broad correlation with damage to the different structures in the liver, and thence to infer the site of disturbance in those illnesses in which pathological manifestations are less pronounced. The next contribution to the interpretation of symptomatology will undoubtedly come from a direct study of the gross functional disturbances themselves.[1]

Before any attempt of analysis of symptomatology may be made, however, one must first bear well in mind what symptoms occur in hepatic insufficiency. "There is commonly a manifestation which has been given too little attention and which indeed has been sniffed at too much, and sniffed of too little. This is the 'fetor hepaticus,' the perception of which often enables one to recognize hepatic insufficiency, even though jaundice may be slight or absent."[2]

Other signs often associated with, but not necessarily indicative of, hepatic insufficiency include palmar erythema, multiple spider-nevi, recent loss of axillary hair, gynecomastia, venous collateral circulation, and hemorrhagic tendency. Ascites and edema are common signs. The size of the liver is often of the utmost significance in prognosis. Rapid diminution is well recognized as one of the most ominous signs of all, indicating diffuse necrosis, and as a rule a fatal outcome, although even here exceptions are noted.[2] Symptomatology includes anorexia, nausea and vomiting, abdominal pain, diarrhea, and easy fatigue. A keen watch should be kept for the slightest sign of undue somnolence, disorientation, or euphoria, since these are often the first and ominous harbingers of hepatic coma.[2]

As has already been seen, there is considerable variance of opinion concerning the relationship between histology, symptomatology, and prognosis. Waldstein et al.[6] have attempted to correlate the major clinical and biochemical findings with the histologic features. These findings, in the majority of cases, may help the clinician predict the pathologic process (except possibly fatty metamorphosis) that is present in the liver and thus aid in the estimation of the stage and activity of the cirrhotic lesion. In keeping with the observations of others, the results indicate that the histologic findings at any one time have little long term prognostic significance. For the time being, prognosis is best judged on the clinical grounds that have been established.

Laboratory Tests: Hanger, of Columbia University, gives the following discussion of laboratory tests and their significance in prognosis of liver disease.[7]

More than 500 functions have been ascribed to the liver. Many of these represent complex enzymatic activities in the intermediary metabolism of foodstuffs and are still beyond the range of clinical study. Others can be measured relatively easily in the human being and thus may be utilized to evaluate the status of hepatic efficiency in health and disease.

Histologic Features[6]	*I Liver Cell Damage*	*II Inflammation*	*III Scarring*	*IV Fat*
Clinical Findings				
Jaundice	x	..	..	..
High jaundice	x	..	..	x
Anemia	x	..	..	..
Ascites	..	x	x	..
Hyperestrogenism	..	..	x	..
Alcoholism	..	..	x	x
Biochemical Findings				
Flocculations	x	x	..	..
Globulins	x	x	..	..
Albumin-globulin ratio	x	x	..	..
Alhumin	x	..	x	..
Total protein	..	..	x	..

The liver may show a wide variety of derangements which may be grouped with certain overlapping as follows: (1) impairment of one or many metabolic functions, (2) abnormalities in the excretion of various constituents of the bile, (3) disturbances of flow of blood through the liver or inadequate distribution of blood to hepatic cells, and (4) inflammation or scarring of the supporting structures of the liver.

It is not unusual to encounter derangements in all these categories during the course of diseases with widespread involvement. On the other hand, lesions of the liver may be highly selective, and, unless the appropriate type of test is employed, the disorder may be completely unrecognized.

Liver function tests are indicated under a variety of clinical circumstances: (1) for the diagnosis of an obvious hepatic derangement such as jaundice or hepatomegaly, (2) to follow the course of a disorder, or (3) for the detection of possible hepatic involvement among subjects who have been exposed to certain infections, noxious agents, or other hazards known to be debilitating to the liver.

In our clinic the following procedures for hepatic investigation are used almost exclusively:

1. For the detection of decreased metabolic activity: (a) total serum cholesterol with the determination of the esterified fraction, (b) serum albumin level, and (c) bromsulfalein clearance.

2. For the recognition of excretory difficulties: (a) total serum bilirubin with the determination of the prompt direct reacting fraction and (b) serum alkaline phosphatase.

3. For the recognition of necrotizing or inflammatory reactions in the liver: (a) cephalin flocculation, (b) thymol turbidity, and (c) serum globulin level.

Every survey should include at least one test from each group.



Thank you doctor for telling mother about...



- The Best Tasting Aspirin you can prescribe
- The Flavor Remains Stable down to the last tablet
- 15¢ Bottle of 24 tablets (2½ grs. each)

We will be pleased to send samples on request

THE BAYER COMPANY DIVISION of Sterling Drug Inc., 1450 Broadway, New York 18, N. Y.

The various patterns of hepatic functional derangement encountered in hepatic disease are determined largely by the acuteness of cellular damage and the extent of involvement. Acute necrotizing lesions usually cause the prompt appearance of a positive cephalin flocculation reaction. The thymol turbidity reaction tends to become positive as inflammatory reactions develop. Both of these tests, therefore, are indicated in the diagnosis of conditions such as acute and chronic hepatitis, active Laennec's cirrhosis, acute anoxia, and cases of poisoning with such agents as phosphorus, carbon tetrachloride, and other noxious agents.

Diets deficient in protein, chronic biliary obstruction, intrahepatic compression, chronic anoxia, repeated exposure to small doses of hepatotoxic agents, alcoholism, etc., lead to gradual damage with diverse manifestations, such as fatty infiltration, hyaline degeneration, atrophic changes, sluggish functioning, and depressed viability of parenchymal cells. Chronic injury often gives rise to selective impairment of hepatic functions, such as lowering of serum albumin, decreased esterified cholesterol fraction in the serum, impaired bromsulfalein excretion, etc., while other functions may be well maintained.

Post and Patek were able to correlate the prognosis of patients treated with a highly nutritious diet with the plasma serum albumin level at the time of the patient's admission to the hospital. On the average, those patients who died had a serum albumin of 2.4 grams per 100 cc. on admission, whereas those who improved and whose ascites disappeared had a serum albumin averaging 3.0 grams on admission, a difference which was statistically significant.[8]

It is obvious that flocculation or turbidity tests, which are usually negative or irregularly positive during the course of sluggish degeneration of hepatic cells, are of little value in the recognition of conditions such as fatty infiltration, chronic passive congestion, neoplastic invasion, and chronic poisoning, unless necrotizing or inflammatory lesions are also present. Further important information may be gained by repeating a small battery of pertinent tests at weekly intervals until the trend of the disease under standardized conditions has been established.

DIETARY FACTORS

The dietetic regimen of patients under Patek's care included approximately 350 grams of carbohydrates, 140 grams of protein, and 175 grams of fat, a total of 3,500 calories a day. The diet consisted chiefly of meat, fish or poultry, milk, eggs, fruit, and green vegetables. The diet was supplemented with a total of 50 grams of powdered brewer's yeast a day, or, if this was not tolerated, an oral B complex preparation. In addition, intramuscular injections of thiamine chloride, 5 mgm. daily, and unconcentrated liver extract, 5 cc. twice weekly, were given.[8, 9]

The intensive combined method of therapy for cirrhosis of the liver used by Morrison and his workers consisted of administering a maximal protein, low fat, high carbohydrate diet in which protein was forced to maximum tolerance by three meat servings and at least six to eight glasses of skim milk and cottage cheese daily. This basic diet was supplemented by daily injections of 5 cc. of whole liver extract fortified with high potency vitamin B complex in large dosage, by oral vitamin B complex and multivitamin capsules, and by two grams each of methionine and choline daily. Such a method resulted in a sharp reduction of mortality, morbidity, and disability rate in the series of patients with chronic hepatitis.[10]

It should be pointed out that the improvement in the prognosis of patients with cirrhosis under the care of Patek and his contemporaries occurred during the era of chemotherapy for infection, and this may account for some of the improvement noted.[8]

The ease with which patients can be induced to eat both the diet and supplementary feedings to a total of 3,500 to 4,000 calories per day is in itself one of the best indices of prognosis. Rapid improvement may be expected in those who will do this with but little urging, and in general the prognosis varies roughly with the appetite, being worse in those with severe anorexia, and, of course, worst in those who are comatose and will eat nothing.

Of crucial importance in all cases with anorexia is the attention given to the total food intake from day to day. All too frequently a sense of false security is gained merely by the fact that an adequate diet has been ordered or that certain amounts of glucose are being given intravenously, without due inquiry as to whether the 24-hour caloric intake has reached a desirable level. It is best that this be maintained on a current basis, from hour to hour. This requires a general awareness of the importance of the problem on the part of the attending physician, house officers, nurses, dietitians, and even the close members of the family, if intelligent and cooperative.[2]

In the work of Eckhardt et al.,[11] three deeply jaundiced patients with active fatty alcoholic cirrhosis were selected for a study of the effect of protein starvation for a short period of time, and of subsequent re-feeding upon their clinical course, liver function, and liver histochemistry. Improvement in clinical status and in liver function occurred in spite of negative nitrogen balance occasioned by diets grossly deficient in or devoid of protein. Liver histology, however, failed to reflect this improvement, and, except for a moderate reduction in liver fat during choline administration, morphologic improvement

COLORFUL COLORADO

invites you to

The Eighth Annual Rocky Mountain Cancer Conference



at Denver
July 14 and 15

No Registration Fee • Headquarters Hotel: Shirley-Savoy

★ There will be an unusually fine program with eight outstanding guest speakers in Radiology, Dermatology, Surgery, Urology, Pathology, Internal Medicine and Gynecology.

★ You will enjoy meeting colleagues from 20 states who annually attend this superb conference.

★ A banquet and special entertainment the first evening will provide relaxation and enable you to enjoy Denver's western hospitality.

★ You can combine attendance at an interesting medical meeting with an enjoyable vacation in the Rocky Mountain West when scenery and climate are at their best.

For information write to
Rocky Mountain Cancer Conference
835 Republic Bldg., Denver 2

SPONSORED BY THE COLORADO STATE MEDICAL SOCIETY AND THE COLORADO DIVISION, AMERICAN CANCER SOCIETY

Plan Now to Be in Denver July 14 and 15

appeared only with the provision of adequate exogenous protein and the maintenance of positive nitrogen balance.

It may be possible, however, to improve further the current regimen of treatment of chronic hepatitis by reducing the work now required of the liver. Energy is required to metabolize amino acids and to form urea from the nitrogen of the molecules. Possibly a decrease in the current high protein intake of 140 to 150 gm. per day might be feasible without a loss in efficacy of the treatment regimen, if lipotropic substances are furnished to the body.[12]

Necrosis and cirrhosis do not respond to the same dietary factors. Once the liver cells are destroyed through the stages of degeneration and necrosis, therapy may only hasten regeneration of new hepatic parenchyma, but it will not achieve direct repair of dead cells. Clinical management should therefore concentrate on the prevention of such serious complications as necrosis, or, if it is already present, on its containment. This may be attempted by a diet high in sulfur-containing amino acids, methionine and cystine, and in tocopherol.[13]

The liver is a junction in the body's transportation system for fat. The causes of the deposition of excess amounts of fat in the liver can be divided into three general groups: first, the mobilization of an abnormal amount of fat to the liver; second, a decrease in the effectiveness of the factors which normally promote movement of fat away from the liver; and third, interference with the normal ability of the liver cells to metabolize fat. It is obvious that under many circumstances more than one of these processes may be operative.[14] If hepatic enlargement is due to fatty metamorphosis or fatty cirrhosis, the prognosis is relatively good; it has become increasingly clear within the recent past that a good diet alone will frequently serve to induce a remission.[2]

Best and his group classify fatty livers on the basis of four general causes: (a) dietary abnormalities, (b) hormonal imbalance, e.g., insulin deficiency, (c) oxygen lack, e.g., anemia or ischemia of liver cells and, (d) toxic or infectious processes. Each type may respond to therapy designed to correct the specific etiological abnormality; there is no single treatment for all fatty livers.[15]

In cirrhosis and diffuse hepatic injuries which are primarily non-fatty and non-dietary in genesis, it is doubtful that dietary therapy is of nearly as great significance in prognosis or treatment. In fact, the more one studies this group, the more one is inclined to the belief that the physician's role is that of protecting the patient against further insult such as may arise from exertion, fatigue, malnutrition, negative nitrogen balance, drugs or chemicals, anesthesia, or needless cholecystectomy or exploration of the common bile duct.[2]

LIPOTROPIC FACTORS

The liver can be made fatty by the addition of an excess of fat to a normal diet. This is, in part, due to a relative deficiency of choline. It can be made extremely fatty by withdrawal of the lipotropic agents from a diet, even if this contains no fat.[14] Fatty infiltration of the liver occurs when the neutral fats cannot be removed from the liver by such transport systems as the phospholipids. The liver is infiltrated with fat whenever the synthesis of phospholipids is impaired. This occurs when there is an insufficiency of essential fatty acids, when there is an insufficiency of choline and methionine, when excesses of cystine cause the utilization of methionine for the formation of protein, and so forth.[8]

The Spanish group has presented interesting data which suggest that the fundamental defect in phospholipid synthesis in hepatic disease is an enzymatic one and not a deficiency of building blocks, since not only was added choline lost almost quantitatively, but also in the presence of hepatic disease a spontaneous cholinuria was present, which subsided with clinical recovery.[16]

In human beings with liver disease, the therapeutic value of choline and methionine has not been definitely established. Choline or methionine is indicated only at the beginning of treatment in patients with fatty infiltration of the liver who are acutely ill and cannot eat. The same lipotropic effect is achieved more slowly without choline or methionine in patients who will eat an adequate diet.[17] While it seems true experimentally that choline exerts a beneficial effect by clearing the fat from the liver, therapeutic trials have not always yielded unequivocal results. The possibility of toxic effects must be taken into consideration. Particularly, the possibility of circulating acetylcholine appears of definite importance.[18]

Hepatotoxic substances such as chloroform. carbontetrachloride, and phosphorus can produce, depending on dosage and duration of administration, fatty changes, necrosis, and fibrosis of the liver. Dietary changes affect the toxicity of such agents. Liver injury may also be increased, probably due to the greater solubility of many of these substances in fat, allowing them a more prolonged toxic action of the compound. Depending on the hepatotoxic agent being used, its dose and route of administration, and the dietary conditions of the study, mortality may be influenced by choline, methionine, cystine, other compounds supplying SH groups, or totally unrelated agents. One would expect supplements of choline to act chiefly with regard to the prevention or alleviation of fatty changes and methionine and cystine to affect the incidence of necrosis.[19]

Further suggestions have been made concerning the application of lipotropic substance therapy. Since



for the 3 patients in 4







with seborrheic dermatitis of the scalp

Have you prescribed SELSUN *for them yet?* Here are the results you can expect: complete control in 81 to 87 per cent of all seborrheic dermatitis cases, and in 92 to 95 per cent of common dandruff cases. SELSUN keeps the scalp scale-free for *one to four weeks*—relieves itching and burning after only two or three applications.

SELSUN is applied and rinsed out while washing the hair. It takes little time, no complicated procedures or messy ointments. Ethically advertised and dispensed only on your prescription. In 4-fluidounce bottles.

Abbott

prescribe...

SELSUN®

SULFIDE *Suspension*

(Selenium Sulfide, Abbott)

405065

ACCEPTED COUNCIL ON PHARMACY AND CHEMISTRY AMERICAN MEDICAL ASSOCIATION

stress, such as anesthesia, trauma, or infection, will induce a decrease in liver function in normal persons, one wonders whether it will prove possible to prevent liver damage by prophylactic administration of lipotropic and other substances when stress can be anticipated.[12]

VITAMINS

Parenteral administration of vitamins totaling several times the daily requirements is advisable.[20] Vitamin K preparations are clearly indicated in the presence of hypoprothrombinemia because of the possibility that defective alimentation is responsible. If reversion to normal with injection of small doses (2 to 4 mg. of the parenteral preparation) does not occur, large doses will probably be ineffective and may possibly be harmful. Thiamine in moderate doses, to aid in metabolism of the added glucose, is clearly indicated.[16] It has been shown that the pyruvic acid level in the blood and spinal fluid rises in direct proportion to the degree of liver failure and that the administration of thiamine does not check this rise.[20]

There is doubt that large quantities of purified vitamins are needed, despite the fact that the liver stores water and fat soluble vitamins. The defect again is probably one of faulty utilization. Niacin in massive doses may be injurious, since it is antilipotropic when excreted, using methyl groups which might otherwise be available for transmethylation or phospholipid synthesis.[16]

Recent work by György with experimental animals indicates that vitamin B_{12} may have strong lipotropic activity in fatty livers.[20]

INTRAVENOUS GLUCOSE

It is believed of value to administer glucose intravenously in any case of hepatic insufficiency in which anorexia is at all prominent, although the mere giving of intravenous glucose should not create the illusion that everything necessary is being done. Should insulin be given with the glucose? Proof of its virtue in hepatic insufficiency has not been provided; nevertheless, it may be beneficial at least in promoting appetite in certain cases.[2]

It should be pointed out, according to Karl et al. in their work with hepatic coma, that several patients who had received large quantities of glucose in water showed a marked drop in blood electrolytes. They had not had paracenteses or mercurial diuretics to account for low salt and potassium levels and must be considered to have had electrolyte dilution on the basis of water intoxication.[20]

It may be inferred that hepatic coma represents a functional disturbance rather than an anatomical one. The "lesion" may be a biochemical disturbance involving carbohydrate metabolism and which perhaps is incidental to a breakdown of certain enzyme systems (cozymase and cocarboxylase), which play a part in the intermediary metabolism of glucose. If these enzyme systems can be sustained and glucose supplied for combusion, there is a possibility of recovery, provided always that sufficient hepatic tissue remains to sustain life.[21]

PARENTERAL PROTEINS

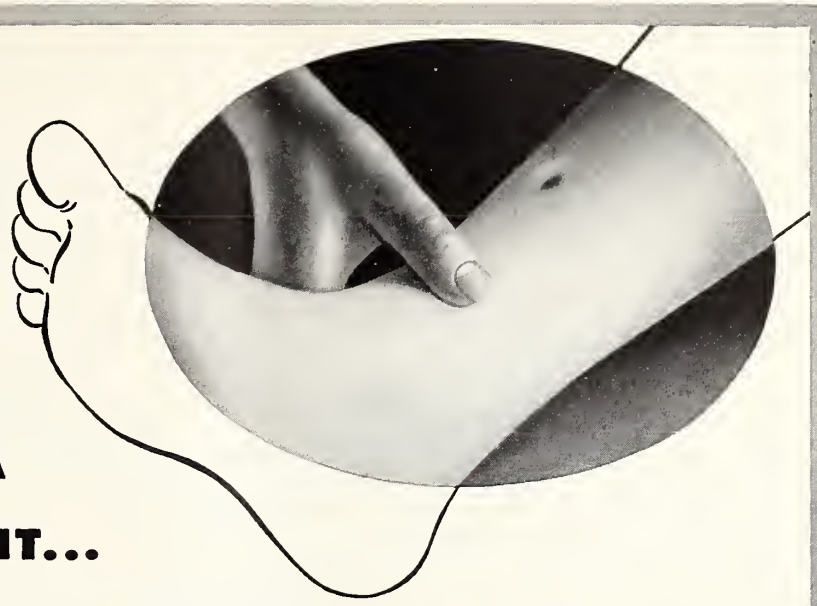
The question often arises whether the patient in coma from acute infectious hepatitis or cirrhosis should be given intravenous amino acids. On theoretical grounds this might be considered dangerous since the sick liver might not be able to metabolize the excess load of amino acids. Actually, however, no deleterious effects have been observed from the intravenous use of commercially prepared amino acids in patients in hepatic coma or ill with severe hepatitis or cirrhosis, although at the same time one would hesitate to ascribe any benefit to them.[8] Karl and his group state that comatose patients with severe liver damage should definitely be supplied proteins parenterally.[20]

Concentrated salt-poor human serum albumin is neither specific for the relief of ascites nor markedly beneficial in the treatment of the underlying liver disease. In view of the variety of results obtained with concentrated human serum albumin in patients with cirrhosis of the liver, it is concluded that other factors in addition to a reduced serum osmotic pressure, as represented by decreased serum albumin concentrations, are active in the retention of fluid.[22] Therefore, it seems reasonably conclusive that serum albumin is of little or no value for producing sustained diuresis, reducing ascites, or improving liver function, and that it may even induce fatal hemorrhage or pulmonary edema.[20]

LIVER EXTRACT

The use of intravenous liver extracts has been recommended for patients with Laennec's cirrhosis or hepatic insufficiency from other causes. Liver extracts were advocated for the treatment of liver disease as early as 1626. Numerous enthusiastic reports of their use appeared in the French literature early in the present century.[8] It is believed that one of the effects of crude liver extract administered in large doses intravenously is to restore and sustain appetite in previously anorexic patients with cirrhosis. Used in conjunction with a full diet, a great nutritional advantage is gained which results in the control of wasting and the restoration of protein storage.[23]

Ralli and her associates, in their work with the administration of large doses of crude liver extract, found the survival in the group treated with liver extract to be significantly more prolonged than in the patients treated in the conventional manner. She states that the fact that the patients receiving liver extract



TO DRAIN THE EDEMA PATIENT...

Effectively • Conveniently...

SALYRGAN®-Theophylline

MERCURIAL-XANTHINE DIURETIC

Solution • Tablets

FOR EDEMA due to cardiovascular and renal insufficiency, as well as hepatic cirrhosis

By a dual action on the kidneys which both increases the volume of the glomerular filtrate and diminishes tubular resorption, Salyrgan-Theophylline rapidly produces copious diuresis.

The response to Salyrgan-Theophylline solution does not "wear out" so that doses may usually be repeated as required, without loss of efficiency.

With Salyrgan-Theophylline tablets taken orally, patients appreciate the gradual, non-flooding diuresis and the greater convenience. Salyrgan-Theophylline tablets "can successfully decrease the patient's burden... either by decreasing the need for frequent mercurial injections or by actually replacing the injections entirely."[1]

WINTHROP

1. Abramson, Julius, Bresnick, Elliott, and Sapienza, P. L.: *New England Jour. Med.*, 243:44, July 13, 1950.

Winthrop-Stearns INC.

NEW YORK 18, N.Y. WINDSOR, ONT.

ACCEPTED COUNCIL ON PHARMACY AND CHEMISTRY AMERICAN MEDICAL ASSOCIATION

Salyrgan, trademark reg. U. S. & Canada, brand of mersalyl

survived longer and were in a better state of nutrition than the patients in the control group suggests that the growth-promoting factors in the liver extract may have been a factor in stimulating regeneration of liver cells. Patek's group, in whom the survival is better than that reported by other investigators, received dried brewer's yeast, which also contains growth-promoting factors.[5] Whatever its action, there is little question of its value in the treatment of liver disease.

ELECTROLYTE AND WATER BALANCE

Numerous agents have been employed in an attempt to control the ascites of hepatic insufficiency, including restriction of sodium intake, mercurial diuretics, and, more recently, the cation-exchange resins. Paracentesis, although often necessary as a last resort, is recognized by all to be an admission of inability to control water balance by any other means. Lowe and Overy state that since rigid dietary sodium restriction spares the patient with hepatic cirrhosis and ascites serious protein loss and the discomfort of frequent abdominal paracentesis, it is considered worthwhile. It is not without hazard, however. Close clinical surveillance is necessary to guard against the serious complication of sodium depletion. If sodium depletion develops, electrolytes should be replaced even if this results in the formation of ascites.[24]

One group reports that seven out of eight patients with hepatic cirrhosis had diuresis with diminishing peripheral edema and ascites following the use of an intravenously administered mercurial diuretic and salt-poor human albumin.[25] Hilton, on the other hand, finds that data indicate that in cirrhotic patients with ascites the renal excretion of potassium following the administration of mercuhydrin may, under certain circumstances, be of such magnitude as to produce hypokalemia and a clinical state indistinguishable in symptomatology from the previously described low salt syndrome. These patients were further characterized by their almost total inability to excrete dietary sodium chloride and their lack of response to mercurial diuretics.[26]

A syndrome indistinguishable from impending hepatic coma has recently been described in patients with cirrhosis of the liver given certain ammonium-containing cation-exchange resins. Elevated blood ammonia levels and an intolerance to certain nitrogenous substances in patients with cirrhosis of the liver suggested that this reaction was related to the ammonium contained in the resin. Untoward effects apparently identical with spontaneously occurring hepatic coma can be provoked in patients with cirrhosis of the liver by the administration of ammonium chloride, diammonium citrate, urea or dietary protein.[27] Further work is required to evaluate the cation-exchange resins fully.

ACTH

The use of ACTH in the treatment of liver disease is still in the experimental stage. Theoretically it should be of value in slowing down the process of fibrosis in the liver. It also seems to be of value in any severe toxic state, though the mechanism is unknown. Colbert et al. found that 5 patients with acute viral hepatitis who received 100 mg. of ACTH daily for periods ranging between 9 and 21 days, showed marked symptomatic improvement promptly. This was associated in all instances with a falling serum-bilirubin concentration. Untoward effects included arthralgia on withdrawal of ACTH, glycosuria, hypertension, edema, ascites, "moon face," and acne.[28]

Of interest in considering the action of ACTH is the work showing that a fat-mobilizing factor appears in the urine of fasting rabbits but is absent when the animals are fed. Since it has been shown that both the growth factor of the pituitary and ACTH exert this fat-mobilizing effect, it will be interesting to determine the nature and physiological actions of the material excreted by fasted animals, and thus learn whether it is an adrenal product or one of the pituitary factors.[14] Further time is needed for the collection of clinical results in the use of ACTH in the treatment of liver disease.

SURGICAL PROCEDURES

In treatment of cirrhosis of the liver, the hepatic artery may be ligated immediately distal or proximal to the gastroduodenal artery without producing necrosis of the liver. Similarly, the splenic artery may be ligated without producing necrosis of the spleen. However, the efficacy of the ligation of the hepatic and the splenic arteries in the treatment of cirrhosis of the liver is questioned. The results obtained in patients operated upon do not warrant its continued usage.[29] Better results have been obtained by making a shunt between the portal vein and the inferior vena cava, but even this is still a last resort procedure.

ANTIBIOTICS

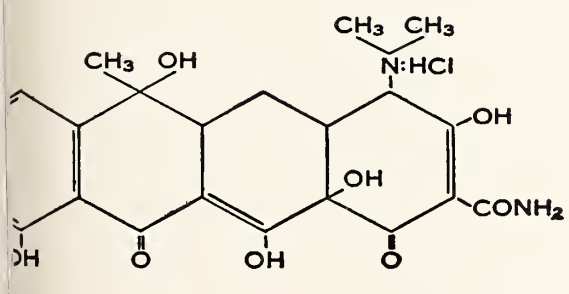
Antibiotics should not be looked upon as substitutes for the established forms of treatment, but rather as adjuncts. After a single intravenous injection of aureomycin, the drug is excreted by the normal liver in high concentration. The ability of the liver to excrete the drug is obviously impaired in the presence of obstructive jaundice or poor liver function.[30] Results with aureomycin to date seem to indicate that it should be given a therapeutic trial in patients with hepatic insufficiency.[31]

Four cases of acute hepatic coma—two secondary to acute viral hepatitis and two to portal cirrhosis—were treated both parenterally and orally with aureomycin. In three cases the improvement was sufficiently

Lederle

MORE RAPID DIFFUSION

FEWER SIDE EFFECTS



LEDERLE LABORATORIES DIVISION

AMERICAN Cyanamid COMPANY

PEARL RIVER, NEW YORK

striking to suggest that aureomycin may well have been responsible for the change, while in the fourth it was difficult to attribute the improvement to any therapy other than the parenteral aureomycin.[32]

If there is reason to suspect from the clinical course or laboratory findings that necrosis is a feature in a patient with coma, it would seem advantageous to administer aureomycin intravenously. Conversely, recent studies have suggested that large doses of aureomycin administered intravenously may actually cause fatty infiltration of the liver.[20] There is evidence that aureomycin administered intravenously to postoperative patients can produce transient clinical icterus, which can be differentiated from icterus from other causes by the observation of its prompt disappearance after the drug has been discontinued.[33]

Aureomycin, when added to a necrotizing yeast diet, has a significant beneficial effect on the prevention of experimental hepatic necrosis in rats. The assumption is made that aureomycin might act through the suppression of the intestinal flora. In the absence of sulfur-containing amino acids or of vitamin E as detoxifying agents, the products of uninhibited intestinal flora may be responsible for experimental acute dietary hepatic necrosis.[34]

ANEMIA

The anemia of liver disease is usually normocytic or macrocytic but may be microcytic when hemorrhage is present. The degree of anemia may be more apparent than real because of an increase in circulating plasma volume without an accompanying increase in total red cell mass. Wintrobe suggests that macrocytic anemia may be due to a variety of causes, such as inability to store hemopoietic principle, deficient diet, and faulty absorption, but that the defect is not related to pernicious anemia. Huang and Wang have suggested that destruction of blood in an enlarged spleen may be a factor. Reports are variable concerning response to vitamin B_{12} and liver extract.[20] Blood replacement therapy may be necessary in the case of extreme hemorrhage, e.g., esophageal varices.

SEDATIVES

By far the greatest danger to patients exhibiting psychotic and motor manifestations of hepatic failure is the indiscriminate use of sedatives. All of the commonly used sedatives, with the exception of bromides and possibly the slowly acting barbiturates, chloral, paraldehyde, and tribromethanol, have been found to exert an unpredictably prolonged and profound effect on patients with grave disease of the liver and commonly precipitate the terminal deep coma in patients who were showing only early evidence of cerebral dysfunction prior to sedation. It seems likely that these drugs in the doses commonly used do not exert any specifically noxious influence on the liver itself, but rather further depress metabolism of neurones gravely damaged by "poisons" or defects in nutrition and impairment of the inactivating functions of the liver.[16]

Analeptics, including glucose, sodium succinate, caffeine, and benzedrine, are without effect upon the comatose state.[35]

SUMMARY

It may be seen that the greatest problem in the treatment of hepatic insufficiency is that the organ affected possesses such reserves of function that it is not until damage is relatively advanced that clinical signs of impairment become apparent. By clarifying the natural history of different lesions, prognosis is placed on a firmer basis. By relating one pathological form to another, the initial lesion of each sequence can be recognized. It is by directing therapeutic and preventive measures against such early stages that the most effective results can be expected.[1]

BIBLIOGRAPHY

1. Himsworth, H. P.: Lectures on the liver and its diseases, Harvard University Press, Cambridge, Mass., 1950.

2. Watson, Cecil James: The prognosis and treatment of hepatic insufficiency, Annals of Internal Medicine 31:3, 405-423 (Sept.) 1949.

3. Capps, R. B., and Norcross, Pliny: Hepatic insufficiency, Medical Clinics of North America 447-459 (Mar.) 1949.

4. Josiah Macy, Jr. Foundation: Liver injury: sixth conference, May 1, 2, 1947.

5. Ralli, E. P.; Leslie, S. H.; Stueck, G. H., Jr.; Shorr, H. E.; Robson, J. S., and Clarke, D. H.: The course of cirrhosis of the liver in patients treated with large doses of liver extract intravenously, Medicine 28:3, 301-330 (Sept.) 1949.

6. Waldstern, S. S.; Popper, H.; Szanto, P. B., and Steigmann, F.: Liver cirrhosis, AMA Archives of Internal Medicine 87:6, 844-862 (June) 1951.

7. Hanger, Franklin M.: Working minimum of hepatic function tests for rationalizing the treatment of jaundice, New York State Journal of Medicine 50:18, 2188-2192 (Sept. 15) 1950.

8. Ratnoff, Oscar D.: Recent concepts in the treatment of hepatic disease, Bulletin of Johns Hopkins Hosp. 84:2, 101-115 (Feb.) 1949.

9. Patek, Arthur J.: Cirrhosis and diet, Journal of the Mount Sinai Hosp. 14:1, 1947.

10. Morrison, Lester M.: Response of cirrhosis of liver to intensive combined therapy, Annals of Internal Medicine 24:1, 465-477 (Mar.) 1946.

11. Eckhardt, R. D.; Zamcheck, N.; Sidman, R. L.; Gabazda, G. J., Jr., and Davidson, C. S.: Effect of protein starvation and of protein feeding on the clinical course, liver function, liver histochemistry of three patients with active fatty alcoholic cirrhosis, Journal of Clinical Invest. 29:2, 227-236 (Feb.) 1950.

12. Action of lipotropic substances in liver disease as measured by radioactive phosphorus, editorial, Journal of the American Medical Assoc. 144:18, 1566-1567 (Dec. 30) 1950.

13. György, Paul: The nutritional aspects of liver injury, Medical Clinics of North America 33:6, 1657-1669 (Nov.) 1949.

14. Best, C. H.: Protection of liver and kidneys by dietary factors, Federation Proceedings 9:2, 506-511 (June) 1950.

15. Best, C. H.; Hartroft, W. S., and Sellers, E. A.: The protection of the liver by dietary factors, Gastroenterology 20:3, 375-384 (Mar.) 1952.

16. Snavely, John Robert: Fatal hepatitis, American Journal of Medical Sciences 219:1, 89-103 (June) 1950.

17. Cuyer and Cornatzer: The use of lipotropic factors in the treatment of liver disease, Gastroenterology 20:3, 385-402 (Mar.) 1952.

18. Dolan, R. A.: Choline and other lipotropic factors; mechanism of action and significance, Minnesota Medicine 31:11, 1198-1205 (Nov.) 1948.

19. Drill, V. A.: Hepatotoxic agents: mechanism of action and dietary interrelationships, Pharmacological Reviews, 4:1, 1-34 (Mar.) 1952.

20. Karl, M. M.; Howell, R. A.; Hutchinson, J. H., and Catanzaro, F. J.: Liver coma, with particular reference to management, AMA Archives of Internal Medicine 91:2, 159-176 (Feb.) 1953.

21. Snell, A. M., and Butt, H. R.: Hepatic coma: observations

ISTINCTIVE AMONG POTENT HYPOTENSIVES FOR THESE

14 DESIRABLE PROPERTIES... Veriloid®

ACCEPTED COUNCIL ON PHARMACY AND CHEMISTRY AMERICAN MEDICAL ASSOCIATION

A selective alkaloidal extract of hypotensive principles obtained by fractionation from Veratrum viride. Representing less than 1% of the whole root, it is freed from the dross of the mother substance. It is generically designated alkavervir. In the management of hypertension it presents these desirable properties.

1 Biologic assay—based on actual blood pressure reduction in mammals—assures uniform potency and constant pharmacologic action.

2 Blood pressure is lowered by centrally medicated action; there is no ganglionic or adrenergic blocking.

3 Therapy is rarely, if ever, fraught with the danger of postural hypotension.

4 Hypotensive action is independent of alterations in heart rate.

5 Cardiac output is not reduced.

6 Renal function, unless previously grossly reduced, is not compromised.

7 Cerebral blood flow is not decreased.

8 Cardiac work is not increased, tachycardia is not engendered.

9 No dangerous toxic effects from oral administration, no deaths attributable to Veriloid have been reported. Side actions of sialorrhea, substernal burning, bradycardia, nausea, and vomiting (due to over dosage) are readily overcome and thereafter avoided by dosage adjustment.

10 In broad use over five years, literally in hundreds of thousands of patients, no other sequelae have been reported, whether Veriloid is given orally or parenterally.

11 Tolerance or idiosyncrasy rarely develops; allergic reactions have not been encountered. Hence tablets Veriloid can be given for the long treatment needed in severe hypertension.

12 Continuing therapy with Veriloid has not led to interference with appetite or with excretory function.

13 Because of its rapidly induced, prolonged action (6 to 8 hours), tablets Veriloid provide around the clock hypotensive effect from 4 doses daily, make today's dosage effective today, and usually prevent hypertensive "spiking" during the night.

14 A notable safety factor in intravenous administration: *extent to which blood pressure is lowered is directly within the physician's control.*

Tablets Veriloid

The slow-dissolving, scored tablets are supplied in 2 mg. and 3 mg. potencies. In moderate to severe hypertension they produce gratifying response in many patients. According to published reports[1] this response can be maintained for long periods in fully 30% of patients; combination with other hypotensive agents has been credited with greatly increasing this percentage.[2] Initial daily dosage 9 mg., given in divided doses, not less than 4 hours apart, preferably after meals. To be increased gradually, by small increments, till maximum tolerated dose is reached. Maintenance dose 9 to 24 mg. daily.

Solution Intravenous

For immediate reduction of critically elevated blood pressure in hypertensive emergencies such as hypertensive states accompanying cerebral vascular disease, hypertensive crisis (encephalopathy), the toxemias of pregnancy. It lowers the blood pressure promptly, to any degree the physician desires, and with notable safety.[3] If excessive hypotensive and bradycardic effects should be invoked they are readily overcome by simple means. Supplied in boxes of six 5 cc. ampuls. The solution contains 0.4 mg. of Veriloid per cc.

Solution Intramuscular

For maintenance of blood pressure in such critical instances, and for primary use in less critical situations which do not show the same immediate urgency. Provides 1.0 mg. of Veriloid per cc. in isotonic aqueous solution incorporating one per cent procaine hydrochloride. A single dose lowers the blood pressure significantly, reaching its maximum hypotensive effect in 60 to 90 minutes. By repeated injections (every 3 to 6 hours) blood pressure may be kept depressed for hours or days if necessary.[4] Supplied in boxes of six 2 cc. ampuls. Complete instructions as to dosage and administration accompany every ampul of the parenteral preparations of Veriloid and should be noted carefully.

1. Kauntze, R., and Trounce, J.: Treatment of Arterial Hypertension with Veriloid (Veratrum Viride), Lancet 2:1002 (Dec. 1) 1951.
2. Wilkins, R. W.: Combination of Drugs in the Treatment of Essential Hypertension, Mississippi Doctor *30*:359 (Apr.) 1953.
3. Stearns, N. S. and Ellis, L. B.: Acute Effects of Intravenous Administration of a Preparation of Veratrum Viride in Patients with Severe Forms of Hypertensive Disease, New England J. Med. *246*:397 (Mar. 13) 1952.
4. Moyer, J. H., and Johnson, I.: Intramuscular Veriloid (Aqueous Solution) As a Hypotensive Agent, Am. J. M. Sc. *226*:477 (Nov.) 1953.

ORIGINAL RESEARCH PRODUCTS OF

RIKER LABORATORIES, INC. 8480 Beverly Boulevard; Los Angeles 48, California



BOOK REVIEWS

Children for the Childless. Edited by Morris Fishbein, M.D. Published by Doubleday and Company, Inc., New York. 223 pages. Price $2.95.

This book consists of a group of topics related to the problem of infertility. The authors are distinguished men in their field, and write with a great deal of authority and experience. The edition has been ably edited by Dr. Morris Fishbein, who also writes one of the chapters.

The book begins with discussion of the sociological problems of infertility and carries through the physiological, psychosomatic, and anatomical problems as related to infertility. There is a special chapter devoted to artificial insemination and its legal problems. The last two chapters review the problem of adoption and discuss some of the genetic factors associated with this problem.

This book can be a valuable adjunct to the physician who is seeing this type of problem. The book is also written so that the average layman could easily understand most of its contents. The chapters on psychosomatic aspects of infertility, adoption, and genetic characteristics of children are exceptionally good. They stress some of the most important problems that are frequently overlooked in this matter. The strictly sociological and scientific aspects of this book are at times confusing and mildly contradictory; only the superficial scientific aspects are covered, but this may be an excellent introduction for anyone interested in this problem.

One noteworthy chapter was written by Dr. Rubin, who was the originator of the Rubin test for tubal patency. His historic review of this subject is enlightening.—*R.H.O.*

The Hepatic Circulation and Portal Hypertension. By Charles G. Child, III. Published by W. B. Saunders Company, Philadelphia. 444 pages, 132 figures. Price $12.

This is a particularly appropriate book for those interested in disease of the liver. The text deals with embryology, anatomy, and physiology of the hepatic circulation, not touching upon the more general areas of physiology and metabolism in which the liver plays such an important part.

Chapter headings such as Comparative Anatomy of the Hepatic Circulation, Embryology of the Liver and its Blood Vessels, The Gross and Microscopic Anatomy of the Intrahepatic Vasculature, The Normal Physiology of the Hepatic Vasculature, The Liver and Shock, Portal Hypertension, Preoperative and Postoperative Water Electrolyte and Protein Balance in Patients with Cirrhosis represent the thoroughness of treatment by the author.

Commendable features of the book are its liberal usage of historical background material, recently accepted concepts of liver anatomy and structure, and excellent documentation of material by experimental evidence and a fine bibliography.

Every student interested in the liver, regardless of his field, will find this book useful.—*M.H.D.*

Salt and the Heart. By E. T. Yorke. Published by Edward T. Yorke, M.D., 1717 North Wood Avenue, Linden, New Jersey. 83 pages. Price $3.45.

The greatest value we find in reading this book is its presentation of an approach to the low sodium diet. The physician who finds it difficult to convince a patient of the value of sodium restriction might gain a lot from these pages. The author opens with a tale of an old sea captain who has developed a heart condition. It describes the various types of treatments used, stressing the low sodium diet and the captain's reaction to it.

The author also discusses briefly some of the symptoms of heart failure and hypertension, the history of the treatment of heart conditions, and the role of sodium in the body. Much of this discussion would be of value to the cardiac patient. However, one is inclined to wonder, while reading the book, exactly what type of reading audience the author had in mind. While much of it would seem to be addressed to the layman, there is considerable treatment of information on determining the levels of dietary sodium desirable in cardiovascular conditions. This type of information certainly has no business in the hands of the layman. Some of the dietary regimes outlined are inadequate in the nutrients recommended by the National Research Council, and one must question the value of urging malnutrition onto these patients. Aside from this, the book is written in an interesting style, and we should stress again its value in teaching the patient.—*M.A.P.*

Surgery of Infancy and Childhood, Its Principles and Techniques. By Robert E. Gross and William E. Ladd. Published by W. B. Saunders and Company, Philadelphia. 1000 pages, 1488 illustrations on 567 figures. Price $16.

This timely and up to the minute successor to *Ladd and Gross' Abdominal Surgery of Infancy and Childhood* is beyond doubt the best source of information concerning pediatric problems which necessitate surgery. The volume can be considered a reliable and complete reference to both the common

BASIC among broad-spectrum antibiotics

true broad-spectrum action
against pneumococci, streptococci,
staphylococci and other
gram-positive and
gram-negative pathogens

unexcelled tolerance

outstanding stability

high blood levels quickly
reached and maintained

may often be effective
where resistance or sensitivity
precludes other forms of
antibiotic therapy

racyn *hydrochloride*

brand of **tetracycline** hydrochloride

Tetracyn Tablets *(sugar coated)*

250 mg., 100 mg., 50 mg.

**English, A. R., et al.: Antibiotics Annual (1953-1954), New York, Medical Encyclopedia, Inc., 1953, p. 70.*

PFIZER Rx

BASIC PHARMACEUTICALS FOR NEEDS BASIC TO MEDICINE

536 LAKE SHORE DRIVE, CHICAGO 11, ILLINOIS

and the unusual surgical conditions of infants and children. Any reader can sense the authenticity of the contents which reflect so much of the personal expeperience of the author, the undisputed leader in his field (2000 to 2500 general surgical operations are performed each year at the Boston Children's Hospital).

The large body of clinical matter is divided into 69 chapters; in most of these each chapter is concerned with a specific type of defect or disease, congenital or acquired. A few chapters deal more generally but pertinently with important aspects of pediatric surgery such as anesthesia for pediatric surgery, preoperative and postoperative care, surgery in premature infants, disruption of abdominal wounds, and rectal bleeding. The illustrations are excellent and clearly explained.

Although the author has not attempted to review the literature on each subject, he refers to pertinent work by others. He "describes for the most part the forms of therapy and management which [his staff] has come to feel are most suitable for the handling of the various diseases and abnormalities" of infants and children.

A complete report of the 95th annual meeting of the Kansas Medical Society, held in Topeka May 2-6, will be published in the June issue of the JOURNAL.

Contributions for Medical Education

A year-end report of the two fund-raising organizations for the nation's 79 medical schools—the American Medical Education Foundation, founded by the A.M.A., and its co-operating agency, the National Fund for Medical Education—was recently issued.

As of December 31, 1953 the American Medical Education Foundation, working through its 53 state and metropolitan committees, recorded a gross return of $1,089,962.93 from 18,176 individual physicians, organizations and laymen. This is the first time that the foundation's annual income has passed the million dollar mark. The number of contributors in 1953 increased 149 per cent over the previous year.

The National Fund for Medical Education, which seeks its funds primarily from business and industry, has just announced that during the past year 994 corporations contributed $1,367,979. This represents an increase over 1952 of 74 per cent in contributions and 193 per cent in the number of participating firms. This record is a tribute to the leadership of 1,000 corporation executives who make up the 47 industry divisions and 17 local sponsoring committees of the Committee of American Industry.

The two fund-raising organizations jointly have distributed nearly $5,000,000 to the nation's medical schools since 1951. Fifty per cent of this money has

PATRONIZE JOURNAL ADVERTISERS

It is advertisers who make possible the publication of the Journal in its present form.

They are deserving of your patronage.

The known clinical advantages of rapid absorption, wide distribution in body tissues and fluids, prompt response and excellent toleration, PROVED by the extensive experience of physicians in successfully treating many common infections due to susceptible gram-positive and gram-negative bacteria, rickettsiae, spirochetes, certain large viruses and protozoa, have established

Terramycin®

Brand of oxytetracycline

as a broad-spectrum antibiotic of choice PROVED

PFIZER LABORATORIES, Brooklyn 6, N.Y.
Division, Chas. Pfizer & Co., Inc.

Pfizer

been contributed by the medical profession and others in the medical field.

Study for Physicians

The average practicing physician, in these busy times, still manages to devote the equivalent of 83.3 eight-hour days a year to keeping abreast of current developments in the field of medicine. This figure is one of many brought out in a preliminary report by the A.M.A.'s Council on Medical Education and Hospitals on its recent survey of postgraduate medical education.

Survey findings are based on data compiled on visits to more than 220 institutions engaged in postgraduate medical education as well as 5,000 questionnaires received from a random sample of 17,000 practicing physicians throughout the country.

Not only are more opportunities for postgraduate work being offered today than ever before, but more doctors are taking advantage of these opportunities. Over 41,000 physicians took some form of postgraduate course last year.

Other highlights of the preliminary report: Some form of organized postgraduate medical course is being offered in every state. . . ..Ninety per cent of these courses are offered in the larger cities. . . . More than 93 per cent of the responding physicians felt that the maximum time they could be away from practice was under 15 days. . . . It cost the average physician surveyed in this study approximately $350 per year to attend postgraduate courses. . . . Chief reason given by those not taking postgraduate courses is lack of someone to care for patients while the doctor is away.

The 103rd annual meeting of the American Medical Association will be held in San Francisco, June 21-25, 1954.

CLASSIFIED ADVERTISEMENTS

FOR SALE—X-ray equipment; complete Profexray, 100 MA 100 KVP, double tube unit. None better made, used very little, practically new, save $1,000. Write the Journal 11-54.

LOCATION WANTED. Will be released from service April 15, will begin residency October 1. Will assist or relieve physician during May, June, July, August. Formerly in general practice. K.U. graduate, member of Kansas Medical Society. Write the Journal 6-54.

FOR SALE—Portable x-ray (Profex), also permanent standard. Other equipment, all in good condition. Write the Journal 7-54.

PHYSICIAN leaving in August or before for one-year residency wants replacement. Small community. Home and office available. Office equipped with EKG, x-ray, all other neccssities. Write the Journal 8-54.

EXCELLENT OPPORTUNITY for man out of internship to gain broad experience with specialist backing. Our group needs a man on a one or two year basis at $1,000 per month. Write the Journal 9-54.

LOCATION—Town of 9,000, 2 colleges, hospital, not overcrowded with doctors, all swell fellows. Modern ground floor office, not much to sell. Partnership if desired. Give qualifications and personal data. Write the Journal 10-54.

THE MEDICAL PROTECTIVE COMPANY
FORT WAYNE, INDIANA
PROFESSIONAL PROTECTION EXCLUSIVELY SINCE 1899
specialized service assures "know-how"
TOPEKA Office:
J. E. McCurdy, Rep.,
1035 Randolph Avenue,
Telephone 2-3027

★ PETRO'S SURGICAL APPLIANCES ★
Surgical Support Fittings by Trained Technicians.
Since 1930, Quality Appliances for Men, Women and Children.
• Abdominal Supports
• Back Supports
• Bras—Breast Forms
• Elastic Hosiery
• Knee Braces
• Ankle Braces
• Taylor Braces
• Trusses
"All Fittings Subject to the Doctor's Approval"
618-20 Quincy TOPEKA, KANSAS Phone 40207

It's Convenient to Buy Kanox

THERAPEUTIC OXYGEN AND ANESTHETIC GASES

Oxygen Therapy

Regulators and Equipment

Repair Service for

Regulators, Flowmeters

and Humidifiers



Nitrous Oxide

Ethylene

Carbon Dioxide

Helium

Oxygen

Cyclopropane

KANSAS OXYGEN, Inc.

Phone 5-5551 HUTCHINSON, KANSAS Box 551

ACCIDENT, HOSPITAL AND SICKNESS INSURANCE

For Physicians, Surgeons and Dentists Exclusively

ALL PREMIUMS COME FROM — PHYSICIANS SURGEONS DENTISTS — ALL CLAIMS GO TO

$5,000 accidental death Quarterly $8.00 $25 weekly indemnity, accident and sickness	$15,000 accidental death Quarterly $24.00 $75 weekly indemnity, accident and sickness
$10,000 accidental death Quarterly $16.00 $50 weekly indemnity, accident and sickness	$20,000 accidental death Quarterly $32.00 $100 weekly indemnity, accident and sickness

Cost Has Never Exceeded Amounts Shown

ALSO HOSPITAL INSURANCE

	Single	*Double*	*Triple*	*Quadruple*
60 days in Hospital	5.00 per day	10.00 per day	15.00 per day	20.00 per day
30 days of Nurse at Home	5.00 per day	10.00 per day	15.00 per day	20.00 per day
Laboratory Fees in Hospital	5.00	10.00	15.00	20.00
Operating Room in Hospital	10.00	20.00	30.00	40.00
Anesthetic in Hospital	10.00	20.00	30.00	40.00
X-Ray in Hospital	10.00	20.00	30.00	40.00
Medicines in Hospital	10.00	20.00	30.00	40.00
Ambulance to or from Hospital	10.00	20.00	30.00	40.00
COSTS (Quarterly)				
Adult	2.50	5.00	7.50	10.00
Child to age 19	1.50	3.00	4.50	6.00
Child over age 19	2.50	5.00	7.50	10.00

$4,000,000.00 INVESTED ASSETS

PHYSICIANS CASUALTY ASSOCIATION
PHYSICIANS HEALTH ASSOCIATION

$19,500,000.00 PAID FOR CLAIMS

51 years under the same management

400 First National Bank Building Omaha 2, Nebraska

$200,000.00 deposited with State of Nebraska for protection of our members

ANNOUNCEMENTS

The 32nd annual scientific and clinical session of the American Congress of Physical Medicine and Rehabilitation will be held September 6-11, 1954, at the Hotel Statler, Washington, D.C. Sessions will be open to all physicians in good standing with the A.M.A. Information may be obtained from the Executive Office, 30 North Michigan Avenue, Chicago 2, Illinois.

The program of the first annual meeting of the Academy of Psychosomatic Medicine, to be held at the Plaza Hotel, New York City, October 8-9, will be devoted to psychosomatic aspects of surgery. Those interested in presenting papers should write Dr. Benjamin Raginsky, 376 Redfern Avenue, Montreal, Canada.

Applications for fellowships and associateships are now being received. New fellows will be inducted at the October meeting.

The sixth American Congress on Obstetrics and Gynecology will be held at the Palmer House, Chicago, December 13-17, under the sponsorship of the American Academy of Obstetrics and Gynecology. Information may be obtained from the Congress, 116 South Michigan Avenue, Chicago 3, Illinois.

Conference on Religion and Psychiatry

In an effort to improve relations between religion and psychiatry, and to encourage the interchange of ideas between representatives of the two groups, eight prominent theologians and nine psychiatrists will meet in Topeka, May 13 and 14. A semi-public meeting will be held the evening of May 13.

Bequest to Clendening Library

A bequest of $1,000 from the late Dr. Edward H. Skinner for the Clendening Library of the History of Medicine of the University of Kansas School of Medicine was announced last month by Dr. W. Clarke Wescoe, dean of the school. Dr. Skinner, who died January 11, 1953, had made other important gifts to the library, including 350 books, some of which are rare and of great value.

Patronize JOURNAL advertisers.

QUESTION?

Have you enrolled in the KANSAS MEDICAL SOCIETY Approved DISABILITY INCOME PLAN?

- **Endorsed and Supervised by Your Society**
- **Non-Cancellable Features**
- **Lifetime Accident Benefits**
- **Five-year Sickness Benefits**
- **30% to 50% Saving in Cost**
- **Over 65% of Eligible Membership Now Enrolled**

Officially approved and endorsed by the Council and the House of Delegates.

For Complete Information Write—Kansas Medical Society, 315 West Fourth, Topeka, Kansas

or

FIRST INSURANCE AGENCY
Washington National Insurance Company
639 New England Building **Topeka, Kansas**

BENADRYL®

Patients troubled by lacrimation, nasal discharge, and sneezing respond to BENADRYL and enjoy symptom-free days and restful nights.

MEDICAMENTA VERA

Parke, Davis & Company

DETROIT, MICHIGAN

TABLE OF CONTENTS

JUNE, 1954

ORIGINAL ARTICLES

Report on the Tubeless Gastric Analysis—R. L. Van Citters, M.D., and Mahlon Delp, M.D., Kansas City, Kansas 311

Multiple Primary Malignant Lesions—Jack W. Welch, M.D., Halstead, Kansas . . 314

Massive Diuresis in Congestive Heart Failure—Samuel Zelman, M.D., Topeka, Kansas . 317

The Use of Hydrocortisone-Neomycin Ointment in the Treatment of Various Dermatoses—Donald L. Fuhrman, M.D., and Bruce V. Drowns, M.D., Kansas City, Kansas 322

EDITORIALS

Chiropractors Withdraw Petitions 325
The New Council 326
Referrals to the Medical Center 327
Thanks, Jackson County 328

MISCELLANEOUS

President's Page 324
95th Annual Session 329
Hemangiomas and Lymphangiomas (Tumor Conference) 338
Committees for 1954-1955 344
Amendments to Constitution and By-Laws . 348
The Anticoagulants: Heparin and Dicumarol—Senior Thesis 352

Entered as second-class matter at the post office at Fulton, Missouri, June 19, 1953, under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of October 3, 1917, authorized on July 2, 1918. Office of Publication: 1201-05 Bluff Street, Fulton, Missouri.

THE JOURNAL OF THE KANSAS MEDICAL SOCIETY

Editorial Board

ORVILLE R. CLARK, M.D., *Editor,* Topeka

JOHN W. CAVANAUGH, M.D., Topeka

DAVID E. GRAY, M.D., Topeka

RICHARD GREER, M.D., Topeka

DWIGHT LAWSON, M.D., Topeka

Associate Editors

DONALD P. TREES, M.D., Wichita

GLEN R. SHEPHERD, M.D., Kansas City

Information

Ownership: The Journal is a non-profit publication owned and published monthly by the Kansas Medical Society.

Subscription: A year's subscription to the Journal is included in membership in the Kansas Medical Society, with $2.00 of each member's dues apportioned to the Journal. Rates to others, except in foreign countries, $4.00 per year or 60c per copy.

Material: Scientific articles, editorials, and data of general interest are invited from all members. Articles are to be submitted on condition that they are contributed solely to this publication. A right is reserved to reject any material deemed unsatisfactory.

Manuscripts: Only manuscripts that are typewritten on one side, double spaced, and original copies can be accepted. Manuscripts will be returned upon request.

Advertising: All advertising contracts, and all copy from advertisers under contract are subject to approval of the editorial board. Copy should be received by the 20th of the month immediately preceding the month of publication.

Non-Responsibility: Although an effort is made to publish only accurate articles and legitimate advertisements, the Journal denies legal responsibility for any statements, opinions, or advertisements appearing under the names of contributors or concerns.

PAULINE FARRELL, Managing Editor and Advertising Manager

OLIVER E. EBEL, Business Manager

THE KANSAS MEDICAL SOCIETY

President.....MURRAY C. EDDY, M.D., Hays
Past President.....LUCIEN R. PYLE, M.D., Topeka
President-Elect.....JOHN M. PORTER, M.D., Concordia
First Vice-President...CLYDE W. MILLER, M.D., Wichita
Second Vice-President, CONRAD M. BARNES, M.D., Seneca
Constitutional Secretary, JAMES A. BUTIN, M.D., Chanute
Treasurer.....JOHN L. LATTIMORE, M.D., Topeka
A.M.A. Delegate, 1954-1955, GEORGE F. GSELL, M.D., Wichita
A.M.A. Delegate, 1955-1956, LAURENCE S. NELSON, M.D., Salina

Councilors and Councilor Districts

First District, FREDERICK E. WRIGHTMAN, M.D., Sabetha
Second District...GLENN R. PETERS, M.D., Kansas City
Third District.....H. PENFIELD JONES, M.D., Lawrence
Fourth District....CHARLES E. VESTLE, M.D., Humboldt
Fifth District, SEVERT A. ANDERSON, M.D., Clay Center
Sixth District.....FLOYD C. TAGGART, M.D., Topeka
Seventh District.....TO BE ANNOUNCED
Eighth District.....JAMES E. HILL, M.D., Arkansas City
Ninth District....LAURENCE S. NELSON, JR., M.D., Salina
Tenth District.....HAROLD M. GLOVER, M.D., Newton
Eleventh District....NORTON L. FRANCIS, M.D., Wichita
Twelfth District.....CYRIL V. BLACK, M.D., Pratt
Thirteenth District..LLOYD W. REYNOLDS, M.D., Hays
Fourteenth District....JUSTIN A. BLOUNT, M.D., Larned
Fifteenth District....ROBERT G. KLEIN, M.D., Dodge City
Sixteenth District.....JAMES L. JENSON, M.D., Colby
Seventeenth District.....H. PRESTON PALMER, M.D., Scott City

OLIVER E. EBEL, EXECUTIVE SECRETARY
RUEBEN M. DALBEC, EXECUTIVE ASSISTANT

Executive Office: 315 West Fourth Street, Topeka

THE JOURNAL
of the
KANSAS MEDICAL SOCIETY

Copyright, 1954, by The Kansas Medical Society

Volume LV JUNE, 1954 No. 6

Report on the Tubeless Gastric Analysis

R. L. Van Citters, M.D., and Mahlon Delp, M.D.
Kansas City, Kansas

In 1950 Segal introduced a method for determining the presence or absence of free HCl in the stomach without subjecting the patient to intubation.[1] Since then his group has published several additional reports,[2, 3, 4] and in the past year other workers have confirmed his findings.

The fundamental principle involved was established by Kelsey and Geiling in 1942 in a publication in which they described a simple quantitative micro method for the determination of quinine in biologic fluids.[5] Segal adapted this procedure to the determination of gastric acidity, using quinine as an exchange-indicator substance. The technique is based upon the simple displacement of quinium ions from an orally administered exchange resin by the action of hydrogen ions present in gastric sections.

$$R\text{-}(\overset{O}{\overset{\|}{C}}\text{-}\overset{-}{O}\text{-}Q\overset{+}{H})_n + n\overset{+}{H}\overset{-}{Cl} \longrightarrow R\text{-}(\overset{O}{\overset{\|}{C}}\text{-}\overset{-}{O}\overset{+}{H})_n + nQ\overset{+}{H}\overset{-}{Cl}$$

Thus, in the presence of free HCl, the quinium ion is released. In vivo it is rapidly absorbed, and about 70 per cent is destroyed by the liver. The remainder may be demonstrated in body fluids, most conveniently in the urine, where about 20 per cent is excreted.

In a given urine specimen the available quinine may be extracted with ether, converted to quinine sulfate, and examined grossly under a suitable ultraviolet light. The amount of quinine present may be directly estimated from the characteristic blue fluorescence of the solution.

In the absence of free HCl, no displacement can occur, hence no quinine is excreted. Actually, experience has shown that small amounts of quinium ion may be released by other cations: Na, K, Mg, and Ca. The concentration of these ions in gastric secretions is extremely limited, consequently only insignificant amounts of quinine appear in the urine because of their action. To further limit their effect, the specimen for examination is collected within two hours of administration of the resin.

PROCEDURE

The following procedure was used. The patient fasts after midnight. A fasting morning urine specimen is obtained and used for control. The patient is then given a secretory stimulant; regular doses of alcohol, caffeine, or histamine have all been found satisfactory. Forty-five minutes later the resin is swallowed with a glass of water. For two hours the patient fasts, does not smoke, and does not void. A two-hour urine specimen is collected, and both this and the control are examined, as follows:

Dilute each specimen to 300 cc. Take a 30 cc. aliquot, make alkaline by addition of 0.5 cc. 0.5 N NaOH, add 15 cc. ether, and shake lightly. Allow the two layers to separate; if necessary, a few drops of alcohol may be added to facilitate separation. Take 8.2 cc. of the ether layer, to it add 5 cc. 0.1N H_2SO_4, and shake lightly. Separate the aqueous layer and examine for fluorescence, comparing with a known solution of quinine sulfate as a standard.

The entire procedure is simple, and only a little practice is necessary to attain proficiency. Further simplification may result from use of a specially de-

This paper, submitted for the March (University of Kansas School of Medicine) issue, was omitted then because of lack of space. Dr. Van Citters and Dr. Delp are from the Department of Medicine, University of Kansas School of Medicine.

signed separatory funnel for the extraction. Standards are easily prepared from commercially available tablets containing the prescribed amount of quinine. A variety of inexpensive ultraviolet lights is available. Any source of approximately 3650 Å at about 4 to 10 watts will suffice.

The resin and stimulant may be self administered and the specimen brought in for examination. The convenience to both patient and physician is obvious. The technique is especially well suited to mass survey for those conditions commonly associated with achlorhydria.

Certain drugs and vitamins which are excreted in the urine may cause fluorescence unrelated to gastric acidity. These include quinine and its derivatives, nicotinic acid, and riboflavin. It is for this reason that controls are run. If there is perceptible fluorescence in the control specimen, the entire determination is void and another test should be attempted a week later.

The present study consists of a series of in vitro procedures demonstrating the properties of the exchange resin, Diagnex*, and a small patient series to confirm its use in a variety of clinical patients.

IN VITRO STUDIES

The following experiments are quantitative determinations of the availability of the quinium ion under varying conditions. For this purpose a fluorophotometer was used, with GE mercury vapor lamp AH-4, primary filter Corning 5860, and secondary filter Eastman "2b." The galvanometer deflections were translated into milligrams of quinine using appropriate tables and standards.

To measure the relationship between pH and the displacement of quinium ion, one patient dose (2 gms.) of Diagnex was incubated in each of a series of buffer solutions of potassium acid phthalate-hydrochloric acid. The solutions were made according to the method of Clark and Lub, and pH was checked on a Weston pH meter. After incubation for 24 hours, the available quinine was measured as quinine sulfate. As demonstrated on the accompanying graph, above pH 3.8 only insignificant amounts of quinine are released. Between pH 3.8 and pH 2, the relationship between pH and quinine released is direct, as evidenced by the straight line graph. Below pH 2, displacement is complete, and all the available quinine may be recovered.

An attempt was made to determine the effect of normal concentrations of cations found in biologic fluids on the displacement reaction. Several doses of Diagnex were incubated in Ringer's solution at pH 7.2, and the quinine concentration was measured. It was established that there is a very small but constant displacement of quinium ion by these cations. No attempt was made to determine the relative displacement by each of the individual cations.

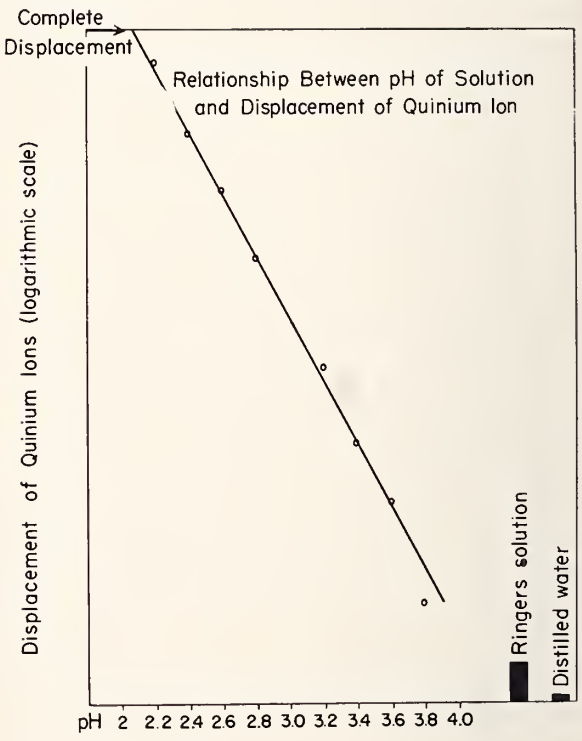
To demonstrate the efficiency of the extraction procedure, samples having known concentrations of quinine and sulfate were quantitatively extracted with the ether-sulfuric acid technique, and the fluorescence was measured and compared with expected values. Results always agreed within 5 per cent. This is in keeping with the work of Kelsey and Geiling[5] and exceeds the accuracy required of this procedure.

Finally, it is essential that a given quantity of available quinine be present in each dose of Diagnex. Any deviation from standard amounts, especially if it be less, could affect the excretion level. Repeated determinations of available quinine failed to uncover any significant discrepancy in the batches from which the study was made.

IN VIVO STUDIES

In vivo studies consist of a small series of patients in whom, for one reason or another, the attending physician found it necessary to determine gastric acidity by the routine fractional method. Although small, the series includes a wide range of acidity and many types of underlying pathology.

In order to demonstrate the effect of ingested fluorescent materials, and to re-emphasize the necessity for running controls, one patient who was found to be achlorhydric by both types of gastric analysis



* Diagnex was furnished by E. R. Squibb and Company.

was placed on a high vitamin diet, and the urine was subsequently examined. There was marked fluorescence. Had this occurred without a control specimen, a false positive would have been reported.

Another patient was found initially to have extremely high values in both control and routine specimen. Closer examination of the chart revealed she had been given a therapeutic dose of quinine several days before. Consequently, it was necessary to wait several days until the control specimen cleared, indicating that excretion of residual medication was complete, until proceeding with the gastric analysis.

Segal has established the excretion of 25 micrograms of quinine as the criteria for acidity, less than 15 micrograms as evidence of anacidity, and the range between as indicative of low acidity, probably requiring that the test be repeated. Although these levels may easily be detected grossly under an ultraviolet light, this series was carried out using the fluorophotometer in addition to the gross inspection method. In this way it was possible to record grossly a positive or negative value for each specimen, and then to measure accurately the actual amount of quinine excreted. These results were used first to check the accuracy of the gross or qualitative method, and second to determine by direct comparison with the values reported in the fractional gastric analysis, the correlation between quinine excreted and the degree of acidity.

From columns 6 and 8 of the following table, it is obvious that no direct correlation exists between degree of acidity and amount of quinine excreted. Two persons whose acidity exceeds 100 units excreted minimal amounts of quinine, while several persons with average acidity excreted large amounts. Note that two persons having less than 20 clinical units of free HCl fell to the middle group of Segal. It is interesting to note that while this series was highly unbalanced by the large number of achlorhydrics used, the test did not fail to identify each of them correctly.

SUMMARY

The tubeless gastric analysis is described in principle and procedure. In vitro tests confirm the dynamics of the test.

A small patient series is included which demonstrates its use in a variety of patients. The test is a simple and reliable means of determining the presence or absence of free HCl in the stomach.

There is no quantitative relationship between the degree of acidity and the amount of quinine excreted.

Number	*Age*	*Race*	*Sex*	*Clinical Diagnosis*	*Fractional Gastric Analysis: Free HCl in Fasting, 15", 30", 45"*	*Free HCl as determined by Gross Fluorescence of Urine*	*Quantitative Excretion of Urinary Quinine Mg.*
1	76	N	M	Ca. Stomach	0-0-0-0	—	.010
2	29	N	F	Anxiety	0-80-112-104	+	.052
3	60	N	F	Osteoarthritis	18-24-52-24	+	.140
4	57	W	F	Involutional Psychosis	0-0-0-0	—	.006
5	42	W	M	Anxiety	0-34-70-70	+	.188
6	67	W	M	HCVD	98-112-120-133	+	.066
7	41	N	M	Anxiety	4-40-36-62	+	.028
8	72	W	M	ASHD	0---78-94	+	.106
9	53	W	F	Diabetes Cirrhosis	4-74-78-52	+	.104
10	58	W	F	Degenerative Bone Disease	38-40-72-72	+	.054
11	61	W	F	Hiatus Hernia	0-46-48-32	+	.044
12	57	W	M	Ca. Pancreas	0-0-0-0	—	.008
13	60	W	F	HCVD ASHD	0-0-100-64	+	.200
14	65	W	F	Pelvic Neoplasm	23-42-42-40	+	.186
15	28	W	F	Multiple Sclerosis	20-60-74-74	+	.194
16	45	W	F	Secondary Anemia	0-0-28-42	+	.178
17	74	W	M	ASHD	0-0-0-0	—	.010
18	64	W	F	Osteo-Arthritis	0-0-4-20	±	.018
19	61	W	M	HCVD	20-60-90-100	+	.046
20	83	W	M	ASHD	30-30-74-88	+	.120
21	34	W	F	Globus	0-0-0-10	±	.020
22	47	W	M	HCVD ASHD	0-0-76-30	+	.037
23	73	W	M	Hernia	0-0-0-0	—	.008
24	72	W	M	Gastric Ulcer	54-64-88-92	+	.044
25	58	W	M	No Diagnosis	0-0-0-0	—	.010
26	41	W	F	Peptic Ulcer	0-38-12-0	+	.036
27	78	W	F	Peripheral Neuritis	32-40-50-52	+	.068
28	66	N	F	Diabetes HCVD	0-0-74-66	+	.060
29	47	N	M	Ca-Stomach	0-0-0-0	—	.008
30	73	W	F	HCVD	0-0-24-32	+	.036
31	53	W	F	Peptic Ulcer	32-40-42-50	+	.128
32	48	W	F	Pernicious Anemia	0-0-0-0	—	.012
33	55	N	M	Pernicious Anemia	0-0-0-0	—	.010
34	50	N	M	Pernicious Anemia	0-0-0-0	—	.006
35	61	W	F	Pernicious Anemia	0-0-0-0	—	.008
36	50	W	M	Pernicious Anemia	0-0-0-0	—	.006
37	34	W	M	Peptic Ulcer	10-20-60-60	+	.084
38	67	W	F	Diabetes	16-58-72-62	+	.044
39	38	W	F	Peptic Ulcer	0-70-94-90	+	.040
40	62	W	F	Arthritis	0-26---20	+	.038
41	51	N	M	Leukemia	34-36-38-42	+	.070
42	45	N	M	Peptic Ulcer	0-14-30-30	+	.116
43	40	W	M	Peptic Ulcer	8-88-94-64	+	.040
44	60	N	M	No Dx.	0-30-42-44-50	+	.064

BIBLIOGRAPHY

1. Segal, H. L.; Miller, L. L., and Morton, J. J.: Proc. Soc. Exp. Biol. and Med. 74:218, 1950.
2. Segal, H. L.; Miller, L. L.; Morton, J. J., and Young, H. Y.: Gastroenterology 16:380, 1950.
3. Segal, H. L.: Med. Clin. N. A. 35:593, 1951.
4. Segal, H. L.; Miller, L. L., and Morton, J. J.: J. Nat. Cancer Institute 13:1079, 1953.
5. Kelsey, F. E., and Geiling, E. M. R.: J. Pharm. and Exp. Therap. 75:183, 1942.

Temperament we are born with, character we have to make; and that not in the grand moments . . . but in the daily, quiet paths of pilgrimage. —*Baldwin Brown*

Multiple Primary Malignant Lesions

Jack W. Welch, M.D.
Halstead, Kansas

Since Billroth first reported the occurrence of multiple primary tumors over 70 years ago, many presentations have subsequently appeared in the American literature. The incidence of multiple primary neoplasms, while not rare, is certainly not commonplace. Warren and Gates[1] report that cases of multiple primary malignant growths represent 3.9 per cent of all cases of cancer. This figure does not differ substantially from the consensus of writers on the subject.

We propose in this paper to present a 10-year summary of the cases of the Hertzler Clinic as well as a partial collective review of the literature and a résumé of a few facets of the problem, including mode of origin and the relation of age, sex, and heredity.

Definition: The postulates laid down long ago by Billroth for the diagnosis of multiple primary cancer are, it is almost universally agreed, far too rigid and difficult of fulfillment. In brief these criteria were that tumors must be in different organs, must be of different cell types, and must produce their own metastasis. We have included in our series only those cases (1) in which there was positive microscopic evidence of malignancy (excepting two cases of cancer of the prostate in which the clinical evidence was overwhelming); (2) in which the tumors presented the accepted and distinct microscopic characteristics of neoplasms from the organs involved, and (3) in which the likelihood of the second or third lesion being metastatic in character has been satisfactorily excluded.

Mode of Origin: The original concept of unicentric origin of malignant tumors, including the division of one cell and growth by geometric progression, has gradually been replaced by that of multicentric origin. The latter concept postulates that malignant tumors are from multiple adjacent foci which coalesce to form the histopathological growth we call cancer. Concrete evidence of the probable truth of the concept is best provided by the carcinoma *in situ*. One of the favorite examples of carcinoma *in situ* is the intraepithelial epidermoid—Bowen's disease—a case of which is included in this series. There is in such a section, graphic evidence of multiple foci of anaplastic change occurring simultaneously; these areas will ultimately join to form a neoplastic tumor. Such areas of carcinoma *in situ* are commonly observed in the cervix, colon (polyp), larynx, breast, and other organs.

Benign as well as malignant tumors are observed to arise from multiple foci. Best known examples of this are the lipomas, the neurilemomas, the pigmented nevi, the keratoses, and the fibromyomata, to mention a few. Occasionally these benign cell clusters undergo malignant change and give credence to the multicentric theory. Notable among these is Von Recklinghausen's disease in which the multiple benign neurofibromas may degenerate into neurogenic sarcomas. Other examples of this include: polyps of the colon, papillomas of the bladder, and myomas of the uterus.

And lastly there is a group of multiple malignant neoplasms which arise from multiple foci; the outstanding example of this is, of course, the lymphoma group. While not conclusive, such evidence strongly suggests the multicentric origin of multiple malignancy.

DISCUSSION

Of the 20 cases finally selected for presentation, only one, Case No. 20, revealed three primary malignant lesions. These three lesions were: adenocarcinoma of the colon, a synovioma of the knee, and a retroperitoneal tumor which proved to be of the lymphoma group; the latter two were quite malignant, of course.

Eleven of the cases were male and 9 were female. This finding is certainly not in keeping with the proportional incidence of male-female found in the literature where the usual observation reveals a predominance of female cases.[2]

The age at the time of diagnosis in our series varied from 38 to 82 with the average age being 63.5. The average age of afflicted females was 60.2, while that of males was 66.6. The average age according to the literature varies from 50 to 60 years. In a series involving so few cases, no definite correlation could be found between age and multiplicity, and none could be found in the various reviews.

In seven of the cases the age of onset and duration of symptoms of the two lesions could not be distinguished as separate occurrences. It does not necessarily follow that the two tumors arose simultaneously, but neither were we able to disprove it. Many instances of simultaneously occuring tumors have been reported.

The possible role of heredity in our small series

From the Hertzler Clinic, Halstead, Kansas.

seems important. In those cases where a reliable family history was available, a significant family history of cancer was obtained in 50 per cent of the cases. This is considerably higher than the average of those reported—between 25 per cent and 28 per cent. Such a percentage should help substantiate the views of many authors that heredity is a definite factor in the etiology of cancer.

The most commonly involved organ in our series was the colon—31.5 per cent. Had multiple skin cancer cases been included in this series, the skin would by far outstrip any other system, for it is the most common site of multiple malignancies. According to Slaughter,[3] carcinoma of the skin is probably the most frequent malignant tumor affecting the human body. This is readily understandable when one considers that the skin is the largest organ system of the body and the one most exposed to carcinogenic agents. By virtue of its size, according to Slaughter, and the extent of its surface area, two tumors may be spaced far enough apart so that there is no possibility of confusing them as one. This is in contrast, he points out, to the cervix, where two separate cell groups undergoing malignant change would necessarily be classified as one tumor.

As regards paired organs diseased by cancer, it is to be expected that the breasts should be the most frequently involved, according to the consensus of writers. Oddly enough, as far as can be determined, a bilateral breast cancer has been seen only twice at the Hertzler Clinic in the last 30 years. In descending frequency, paired organs involved by cancer are: ovaries, testicles, salpinges, and kidneys—none of which are included in this series. The only case of bilateral involvement was that of Case No. 16, where a 68-year-old female was found to have bilateral thyroid malignancy. The left lobe of the thyroid proved to be a nodular colloid goiter with small Hürthle cell tumor, while the right lobe presented an hemangio-endothelioma. Involvement of both lobes of the thyroid with one malignancy is uncommon—both surgical pathological and autopsy examination reveals unilateral involvement except in rare cases.

Slaughter[4] reports three cases of bilateral simultaneous thyroid carcinoma, but nowhere in the literature was there found a case of simultaneous bilateral involvement with two such unsual malignancies. The hemangio-endothelioma of the thyroid is indeed an unusual tumor in America, only three cases thus far having been reported in the literature. In Switzerland, on the other hand, 20 per cent of thyroid malignancies are hemangio-endotheliomas. Thyroid adenomas are not infrequently bilateral, and the incidence of malignant change in them is high enough to dictate removal when possible. Even so, bilateral malignancy is a rarity although, as Slaughter points out, it probably should not be, because of the centrifugal venous and lymphatic drainage coupled with the incidence of bilateral adenomas.

Another case of particular interest to us was that of Case No. 17, where the patient was afflicted with a lymphoma as well as malignant melanoma. This case, too, in our review of the literature, proved to be a rarity. The malignant melanoma occurred first, symptomatically and surgically, and an unusual feature of the case has been the length of time the patient has remained not only alive but well and asymptomatic with at least two highly malignant lesions. The patient first noted an enlarging black mole on his face in 1942. Between 1942 and 1947, he several times shaved the lesion off his face, and three times had it cauterized. In 1947, five years prior to our biopsy, he had had a positive microscopic diagnosis of malignant melanoma by a reliable microscopist. Since then, the diagnosis has been substantiated by the Hertzler Clinic, the Mayo Clinic, and the University of Kansas Medical Center. At the latter institution a further biopsy revealed the lymphoma, which we have confirmed. This man, when last seen, was well and asymptomatic, except for lymphadenopathy, 5 to 10 years after onset. It has been said that malignant melanomas metastasize before the surgeon lays down his scalpel, and death follows not long thereafter. This case proves to be an exception.

Case No. 13 is that of two separate and unrelated carcinomas within the abdominal cavity. Hanson and Crone[5] reported a case where two such malignancies, a papillary cystadenocarcinoma of the ovary and an adenocarcinoma of the sigmoid colon, occurred simultaneously. They further report that in 370 cases of multiple primary carcinoma in a 10-year period at Memorial Hospital, New York, no such case was found. In Case No. 13, here presented, the two tumors were removed surgically within a two-year period, but the likelihood of their being simultaneous is remote.

REVIEW OF CASES

Slaughter[6] presents a collective review of the literature showing 1,868 cases of multiple primary malignancy. Table 1 reveals the distribution of his collected cases in major groups. Table 2 is taken from Barrett[7] et al. and shows the reported incidence of multiple primary malignant tumors. Table 3 represents our own series of multiple malignancies for comparison.

Stalker[8] et al., in their review of 2,500 cases of malignancy in 1937, found 113, or 4.52 per cent, were multiple. Schreiner and Wehr,[9] in 1934, found multiple primary malignancies in 307 of 11,212 cases of cancer for a 2.7 per cent incidence. Bugher,[10] in 1934, reported 30 cases out of 983, or a 3.1 per cent occurrence rate. He computed the anticipated occur-

rence of coincidental multiple malignancies from the cancer statistics of the United States in an eight-year period and found that it was exceeded by the actual occurrence. And according to Bacon and Goss,[11] "Multiple tumors occurring in different organs suggest a tumor diathesis inasmuch as they occur more frequently than could be expected on the basis of chance alone."

Ettinger et al.[12] report a case of six separate primary carcinomas in one patient; these involved, respectively, the jejunum, transverse colon, sigmoid colon, rectosigmoid colon, urinary bladder, and skin. This certainly must represent tumor diathesis. They conclude, "Actually the occurrence of one or more additional primary neoplasms in a patient already afflicted with one such lesion is six or seven times as likely as the first occurrence of a malignancy in an unafflicted individual."

TABLE 1

THE DISTRIBUTION OF MULTIPLE TUMORS—MAJOR GROUPS

	No. of cases	*Percentage*
Multiple carcinomas of the same organ	749	40.0
Multiple carcinomas of varied organs	269	14.4
	1,018	54.4
G. I. tract, multiple carcinomas, from mouth to anus	145	7.7
G. U. tract, multiple carcinomas	18	.9
Female genitalia, multiple carcinomas	32	1.7
Carcinomas of skin associated with G. I. tract	51	2.7
Carcinomas of skin associated with G. U. tract	55	2.9
Carcinomas of skin associated with breast	39	2.1
Carcinomas of breast and G. U. tract	65	3.4
Carcinomas of breast and G. I. tract	35	1.9
Carcinomas of G. U. and G. I. tracts	120	6.4
Miscellaneous multiple carcinomas	104	5.6
Carcinoma of lung with other cancers	34	1.8
Sarcomas, multiple in same organ	7	.3
Sarcomas, multiple in paired organs	16	.6
Sarcomas, multiple in different systems	3	.2
Sarcomas, associated with carcinoma in the same organ	35	1.8
Sarcomas, associated with carcinoma in symmetrical organ	5	.4
Sarcomas associated with carcinoma in same system	4	.3
Sarcomas miscellaneous	82	4.3
	1,868	

Taken from Slaughter, D. P., International Abstract of Surgery 79:92, 1944.

TABLE 2

REPORTED INCIDENCE OF MULTIPLE PRIMARY MALIGNANT TUMORS

Source	*Rate Per Cent*
Second Budapest Pathological Hospital (7)	0.3
University of Milan (7)	0.5
Erisman General Hospital, Leningrad (7)	1.87
Schreiner and Wehr	2.7
Hurt and Broders	3.34
Lichtman	3.50
Warren and Gates (32)	3.70
Stalker, Phillips, and Pemberton	4.52
Berson and Berger	4.60
Owen	4.70
Warren (31)	6.00
Warren and Erhenreich (27)	6.8
United States Average (32)	3.90
Combined European and United States Average	1.84

CONCLUSIONS

1. There is strong evidence in favor of the multicentric origin of multiple malignant tumors.

2. It is fairly firmly established that one primary malignant tumor does not inhibit the development of another.

3. There is good evidence to confirm a familial tendency in the development of malignancy whereas the relation of age and sex is inconclusive.

4. An individual who has developed one cancer is more prone to develop a second than an unafflicted person is to develop the initial one.

5. Multiple malignancies occur more frequently than could be expected on the basis of chance alone.

6. The latter two facts should be recognized, thereby affording the patient the possibility of another surgical cure and avoiding the erroneous diagnosis of metastasis.

7. A high index of suspicion, compatible with the incidence of multiple malignancy, should be maintained always.

8. And, lastly, the cancer returnee should have more than a routine checkup for recurrence; a

TABLE 3

Case	*Age*	*Sex*	*Duration*		*Site*		*Family History*
1	68	F	3 mo.	1 yr.	cervix	lung	negative
2	65	F	14 mo.	14 mo.	pancreas	lung	++
3	57	M	1 yr.	9 mo.	lip	salivary gland	negative
4	74	F	3-4 yr.	3-4 yr.	skin	nose	++
5	69	M	3-4 yr.	9 mo.	rectum	colon	+++
6	82	M	6 mo.	2 yr.	lower sigmoid	transverse colon	negative
7	68	M	1 yr.	1 yr.	prostate	lung	+
8	77	M	1-5 yr.	6 mo.	foot	ear	incomp.
9	75	F	3 yr.	3 yr.	cecum	breast nipple	negative
10	52	F	7 mo.	4 yr.	R. E. system	skin	negative
11	51	F	5 mo.	1 mo.	cervix	urethra	+
12	77	M		6 mo.	prostate	submax. gland	+
13	38	F		6 mo.	rectum	cervix	incomp.
14	52	F	2 yr.	4 mo.	sigmoid	ovary	negative
15	72	M	4 mo.	4 mo.	rectum	recto-sigmoid	incomp.
16	67	F	14 mo.	14 mo.	thyroid	thyroid	+
17	48	M	5 yr.		melanoma skin	R. E. system	negative
18	56	M	6 mo.	3 mo.	colon	lung	negative
19	70	M	2-3 mo.	2-3 mo.	sigmoid	rectum	+
20	57	M	9 mo.	18 mo.	colon	R. E. system	incomp.

thorough periodic search for a possible second malignancy should be made a part of the examination.

SUMMARY

1. A selected and somewhat unusual series of 20 cases of multiple primary cancer treated at the Hertzler Clinic in the last 10 years has been presented.

2. A few of the facets of the problem of multiplicity of tumors have been discussed, including mode of origin and the relation of age, sex, and heredity.

3. A brief and partial review of the literature has been assayed.

4. A few conclusions, drawn from the literature and from this series of cases, have been presented.

BIBLIOGRAPHY

1. Warren, L., and Gates, O.: Multiple primary malignant tumors, Am. J. Cancer 16:1358, 1932.
2. Stalker, L. K.; Phillips, R. C., and Pemberton, J. de J.: Multiple primary malignant lesions, S. G. and O. 68:600, 1939.
3. Slaughter, D. P.: The multiplicity of origin of malignant tumors, International Abstract of Surgery 79:93, 1944.
4. Ibid. pg. 96.
5. Hanson, K. B., and Crone, J. T.: Simultaneous occurrence of multiple primary carcinoma within the abdominal cavity, American J. of Surgery 74:896, 1947.
6. Slaughter, D. P.: The multiplicity of origin of malignant tumors, International Abstract of Surgery 79:92, 1944.
7. Barrett, W. D.; Miller, K. T., and Fessenmeyer, C. R.: Multiple primary cancer, S. G. and O. 89:768, 1949.
8. Stoeken, L. K.; Phillips, R. B., and Pemberton, J. de J.: Multiple primary malignant lesions, S. G. and O. 68:595, 1939.
9. Schreiner, B. F., and Wehr, W. H.: Am. J. Cancer 20:419, 1934.
10. Bugher, J. C.: Am. J. Cancer 21:809-824, 1934.
11. Bacon, H. E., and Goss, O. C.: Am. J. Surgery 69:246, 1945.
12. Ettinger, J.; Massel, T. B., and Wilkins, F. B.: The Am. J. Surgery 78:897, 1949.

Massive Diuresis in Congestive Heart Failure

Samuel Zelman, M.D.
Topeka, Kansas

The current mode of therapy of congestive heart failure, including bed rest, restriction of sodium intake, adequate digitalization, and mercurial diuresis, commonly provides gratifying results in most cases and occasionally allows the physician to witness truly remarkable effects. An instance of such massive diuresis was recently observed, with weight loss amounting to 67 pounds in 10 days, transforming the patient almost unrecognizably, and resulting in no ill effect upon the body economy. That such massive diuresis may carry its hazards, however, is illustrated by another patient who lost less weight over a longer period (62.5 pounds in 21 days) yet developed the low salt syndrome, although therapy was little different in the two cases.

Case 1. Massive diuresis (67 pounds in 10 days) in congestive heart failure, resulting in no ill effect.

A 63-year-old unmarried white male was admitted to the medical service of Winter Veterans Administration Hospital on January 29, 1953, complaining of swelling of the body of six weeks duration. He had been in this hospital in February and March, 1952, complaining of shortness of breath and cough, and he was then found to be in congestive heart failure secondary to arteriosclerotic and hypertensive heart disease; he had been digitalized with relief of symptoms and control of failure, and he had been advised to continue taking digitoxin 0.2 mgm. daily. However, he had taken only an occasional tablet at long intervals. He continued well, nevertheless, until approximately six weeks prior to the present admission, when his ankles began to swell. The legs became swollen in turn. In the final two weeks before hospitalization, the scrotum and penis were swollen, and in the past week the abdomen had become enlarged and firm.

During this development, the patient had continued his usual occupation of sitting under a tree. He had noted no cough or orthopnea, but had been short of breath on exertion. Nocturia had been present two or three times during the past few weeks. The patient had measured this in a dipper, estimating it to equal his intake and concluding, therefore, that he remained in good health. In the past week, he had consulted two different physicians, both of whom diagnosed congestive heart failure and advised hospitalization. This unanimity of opinion brought him to us. He had taken 3 or 4 digitoxin tablets during the past month.

On examination, he appeared obese and markedly edematous, moving with difficulty but otherwise appearing normally alert and not acutely ill. He did not cough, but was orthopneic and dyspneic on slight exertion. The oral temperature was 98.6 degrees F. The conjunctivae were congested and moderately chemotic. The ocular fundi showed narrowed and tortuous arterioles with accentuated bright light streaks and well marked venous compression; there was no edema of the discs. Diffuse retinal degeneration, pigmented and hyaline, was more marked on

From the Medical Service, Winter Veterans Administration Hospital.

the right, but there were no recent exudates or hemorrhages. There was evidence of recent epistaxis in the right nostril. Many teeth were absent, and the remainder were in poor condition. The neck veins were markedly engorged and showed auricular pulsations.

Respiratory excursions of the chest were limited equally on the two sides, but the respiratory rate was not increased. A surgical scar in the left axilla represented empyema drainage 23 years earlier. The lung fields were clear to percussion, but on auscultation there were moist rales at both lung bases posteriorly. The heart was transverse in position, the apical impulse poorly localized but displaced downward and to the left. There were no murmurs. The pulse was regular at a rate of 96 per minute, and the blood pressure was 190 mm. systolic and 120 mm. diastolic. The arterial pulses were intact, and there was no tenderness over veins.

The abdomen was greatly distended and unusually firm, with bulging flanks which were dull on percussion. Palpation was not possible, but there was no tenderness, rigidity, or hernia. There was marked edema of the genitalia. Tinea cruris was present. Marked edema of the legs and thighs extended up the back to involve the posterior chest wall. The color and temperature of the skin were normal. There were senile keratoses of the face and chest.

On the morning after admission, urinalysis revealed no abnormality other than an occasional hyaline cast. Hematologic study showed 4.92 million red blood cells and 4800 white blood cells per cubic millimeter. The hematocrit was 47 per cent. The differential white cell count was not unusual. The hemoglobin was 14.1 grams per cent. The sedimentation rate was not increased. Serologic tests for syphilis were negative. Roentgenogram of the chest showed marked cardiac enlargement, with transverse diameter of 21 cm. and pulmonary congestion (Figure 1). Partial collapse of the lower left lung field had been present on previous films, due to thoracoplasty for empyema 23 years earlier. Electrocardiogram revealed a PR interval of 0.23 second, left bundle branch block, nodal and ventricular extrasystoles; it was essentially unchanged from the tracings obtained on previous admissions.

The patient was digitalized with 1.2 mgm. of digitoxin on the day of admission, followed by three daily doses of 0.4 mgm. before reduction to a maintenance dose of 0.2 mgm. per day. Mercuhydrin (meralluride sodium) in 1 cc. dosage was administered intramuscularly daily for the first seven hospital days, and none was used thereafter. An initial Karrell milk diet (800 cc. milk per day) was changed after two days to a high protein 1500 calorie diet, low in salt. The patient responded with a marked and continued diuresis, losing 47.5 pounds in the first three days, 62.5 pounds in the first seven days, and a maximum of 67 pounds in 11 days, subsequent-

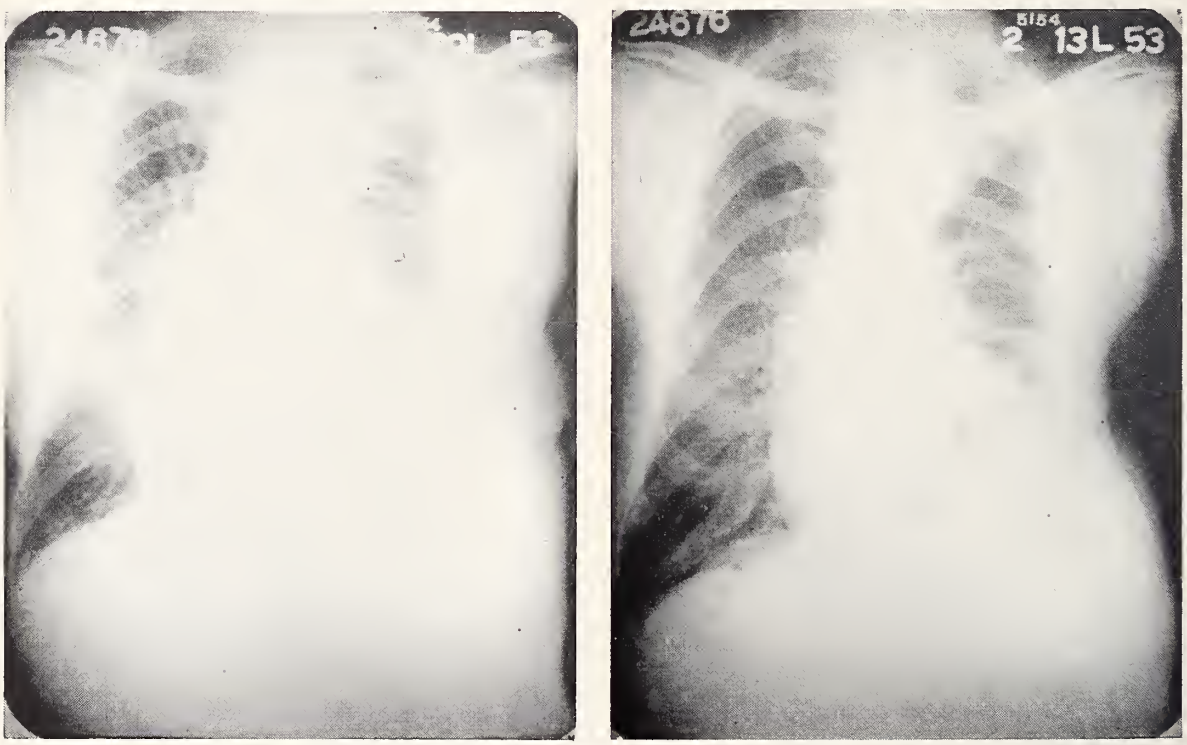


Figure 1. Case 1. Roentgenograms of the chest, posteroanterior view, 6-foot focal-film distance. Transverse cardiac diameters (left) January 29, 21 cm., (right) February 13, 17 cm.

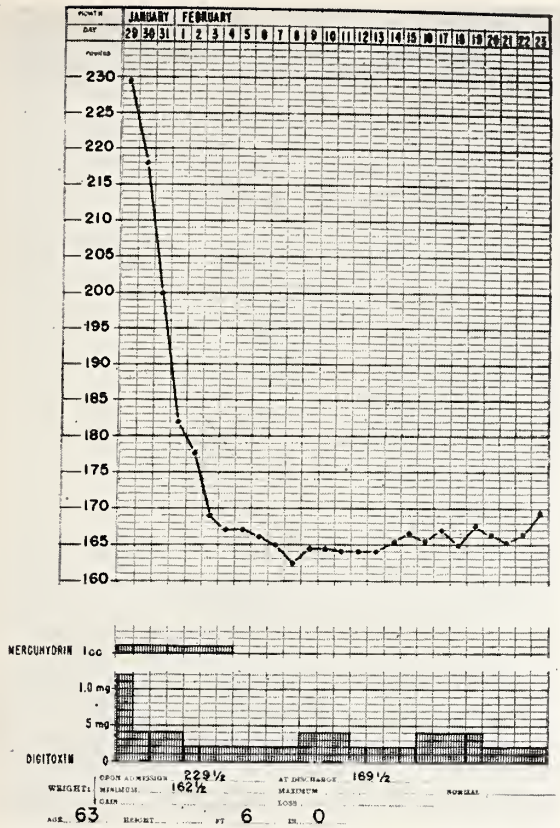


Chart 1. Case 1. Massive diuresis (67 pounds in 10 days) in congestive heart failure, resulting in no ill effect.

ly regaining 4 pounds over the following two weeks (Chart 1). In the first two hospital days (actually only 40 hours), the patient's urinary output exceeded his intake by over 13 liters, during which time he lost 29.5 pounds in weight. Subsequently, recorded urinary output failed to account for actual weight loss, due to the patient's lack of full co-operation after being granted bathroom privilege.

The lung bases were clear of congestive rales on the second hospital day. The next day, dyspnea and orthopnea had subsided. Edema was much less, and the neck veins were much less congested. Anorexia, present on admission, had disappeared, and the patient was hungry. The heart was much smaller, with a widely transmitted mitral systolic murmur not previously observed. A large epigastric hernia, 6 cm. in diameter, was also unmasked by the subsiding edema. On the fourth hospital day, the neck veins were collapsed, and digitalis was reduced to maintenance dosage of 0.2 mgm. daily.

Blood electrolyte studies were done February 3, on the sixth hospital day, after a loss of 60.5 pounds in weight. There were no symptoms suggestive of electrolyte imbalance, the study being prompted only by the observed marked diuresis. On this day, urinalysis revealed occasional casts and a faint trace of albumin. The total and differential white blood cell counts were normal, but the red blood cell count had risen to 5.64 million per cubic millimeter, with a hematocrit of 56 per cent and hemoglobin of 17 grams per cent. The sedimentation rate (uncorrected) had dropped from 7 to 5 mm. per hour (Wintrobe method). Serum sodium was 141.3 and potassium 4.5 mEq/L. The serum chlorides were 89 mEq/L. and the carbon dioxide combining power 68 volumes per cent. The blood urea nitrogen was 18 mgm. per cent. Three days later, the serum chloride level was still 90 mEq/L. and was not again determined. Subsequent blood counts, as late as February 18, the 21st hospital day, continued to show unchanged red blood cell, hemoglobin, hematocrit, and sedimentation values, suggesting these to be the patient's normal figures rather than representing hemoconcentration as originally supposed. The initial blood study presumably reflected dilution by the increased plasma volume of congestive heart failure.

The patient's appearance of edema and obesity revealed itself to be wholly due to edema, as he metamorphosed into a tall, thin, elderly gentleman of distinguished appearance. Because this remarkable change was unanticipated, photographs were, unfortunately, not obtained. On February 4, his calorie restriction was removed. Despite continued digitalization and two subsequent brief periods of increased digitalis dosage, as indicated in Chart 1, he did not become fully compensated. The heart size on chest x-ray, though reduced markedly, remained enlarged with a transverse diameter of 17 cm., and the arm to tongue circulation time did not fall below 19 seconds. He was discharged with urgent advice to continue salt restriction and daily maintenance dosage of digitoxin, and he presumably returned to contemplation of the passing scene from under his favorite tree.

Case 2.* Massive diuresis (62.5 pounds in 21 days) in congestive heart failure, followed by electrolyte disturbance.

A 58-year-old single Negro male window cleaner was admitted to Winter Veterans Administration Hospital May 24, 1953, in obvious congestive heart failure. He had been at this hospital previously, twice in 1948 and twice in 1951, all four times with bronchopneumonia complicating pulmonary fibrosis and emphysema, secondary to chronic bronchial asthma, and all four times responding rapidly to penicillin therapy. At no previous admission had heart failure been recognized, but review of the chest roentgenograms revealed cardiac enlargement amounting to 2 cm. in transverse diameter in May 1951, with reduction to normal size within two weeks. Dyspnea, at first only on exertion, later at rest, had steadily in-

* This case is reported with the permission of Dr. Harry P. Gray of the Medical Service.

creased during these five years, while the pulmonary fibrosis visualized on roentgen films showed steady progression. Clubbing of the fingers had been present since the first observation in 1948.

On this admission, the patient stated that increased dyspnea, orthopnea, and swelling of the legs had been present for the past three months, and that more recently the abdomen had become tightly swollen. He had continued to attempt ambulation on crutches, but he had fallen several times and so turned in to hospital.

On examination, he appeared acutely ill, dyspneic at rest, orthopneic, cyanotic, and mentally confused. His temperature was 100.0 degrees F., the pulse regular at a rate of 104 per minute, and respirations were 48 per minute. The blood pressure was 160 mm. systolic and 90 mm. diastolic, somewhat increased over that of previous admissions. Bilateral arcus senilis was present. Funduscopic examination revealed widened arteriolar walls with atheromatous patches and venous compression. The neck veins were markedly distended.

The chest was of asthenic type, nearly fixed in inspiration, with moderate rounded dorsal kyphosis and increase in anteroposterior diameter. Fine, medium, and coarse moist rales were present throughout the chest. The maximal apical impulse could not be localized. The heart beat was rapid and regular, with a diffusely transmitted systolic murmur and reduplicated second sound at the base. The liver was palpable 10 cm. below the costal margin, and was smooth, firm, and tender. The abdominal wall, genitalia, back, and legs were markedly edematous.

Blood studies on admission revealed 5.09 million red cells per cubic millimeter, and 15.0 Gm. hemoglobin per 100 cc., with a hematocrit of 48 per cent and sedimentation rate (uncorrected) of 9 mm. per hour (Wintrobe method). Serum proteins were 3.2 Gm. per cent albumin and 3.7 Gm. per cent globulin. Blood urea nitrogen was 27 mgm. per cent, and serum chlorides 90 mEq. per liter. Urinalysis revealed a specific gravity of 1.010, two plus albumin, 6-7 red cells and numerous white cells per high power field, and occasional hyaline casts. Blood serology showed 32 Kolmer units, a negative qualitative Kahn, and V.D.R.L. test positive in two dilutions. Roentgenogram of the chest revealed marked cardiac enlargement and pulmonary congestion, not present on earlier films (Figure 2).

Therapy was begun immediately on admission, with oxygen by nasal catheter, digitoxin, mercurial diuretics, and a low salt diet. The daily doses of digitoxin and mercuhydrin (meralluride sodium) are indicated in Chart 2, which graphically indicates the patient's diuretic response. This was promptly initiated and progressed steadily, though not as rapidly as in Case 1, until loss of 62.5 pounds had occurred in 21 days. Symptomatic and clinical improvement occurred concomitantly. By the eighth day, edema was present only below the knees and four days later was nearly gone, and the patient was ambulatory. Urinary outputs were not accurately recorded, and so

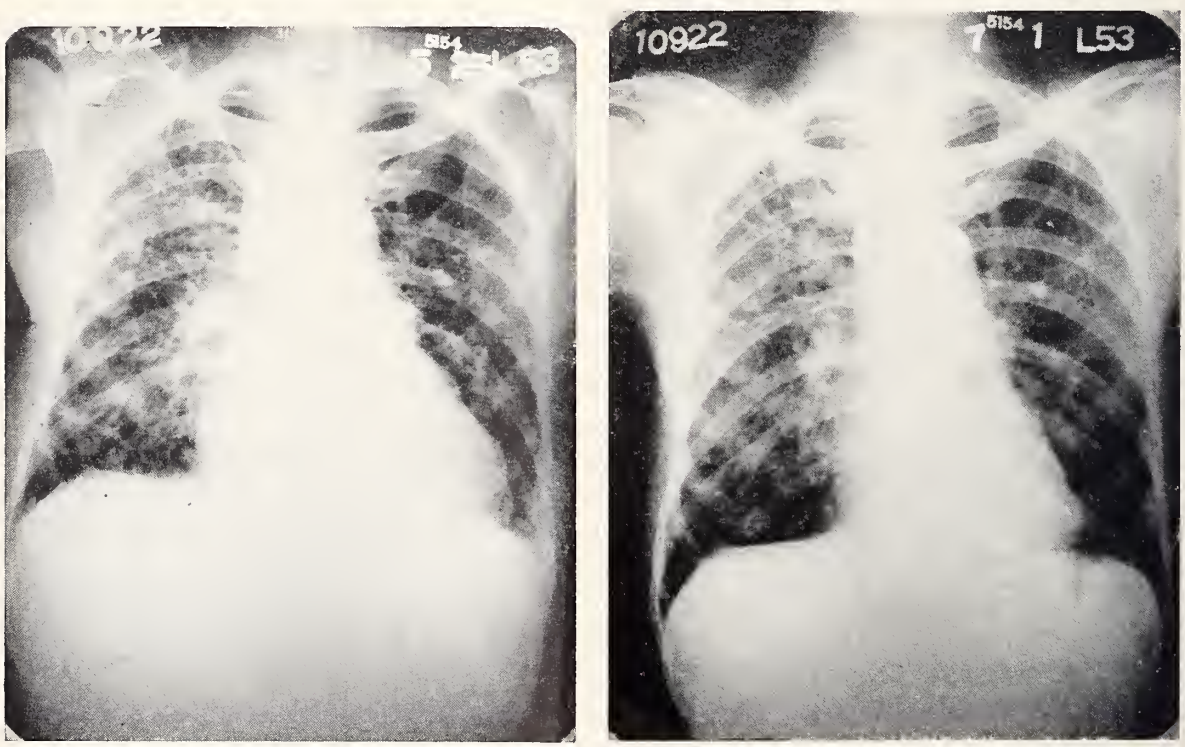


Figure 2. Case 2. Roentgenograms of the chest, posteroanterior view, 6-foot focal-film distance. Transverse cardiac diameters (left) May 25, 18.5 cm., (right) July 1, 14.5 cm.

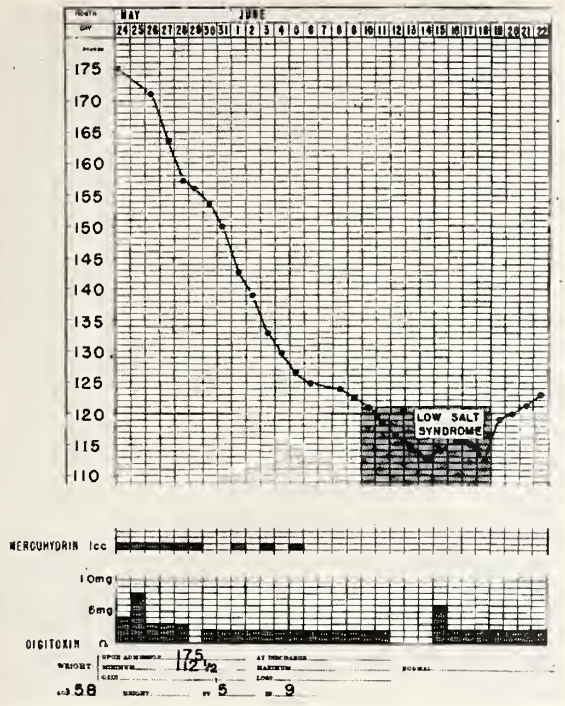


Chart 2. Case 2. Massive diuresis (62.5 pounds in 21 days) in congestive heart failure, followed by electrolyte disturbance.

are not included in Chart 2. The transverse cardiac diameter on chest x-ray was reduced from 18.5 cm. on May 25 to 15.5 cm. on June 9 and 14.5 cm. on July 1. On earlier chest roentgenograms of 1948 and 1951, the diameter had been 12.5 and 13 cm.

The patient's gratifying improvement was interrupted by the appearance of anorexia, nausea, vomiting, diarrhea, weakness, and drowsiness between June 10 and 18, the 18th and 26th hospital days. Atropine medication during this period proved of little benefit. Discontinuation of digitoxin therapy for three days was similarly unavailing. Blood urea nitrogen on June 9 had been 33 mgm. per cent and on June 16, 37 mgm. per cent. The hematocrit on June 9 was 60 per cent, with 6.31 million red blood cells per cubic millimeter and 18.3 Gm. hemoglobin per 100 cc. of blood, the sedimentation rate having dropped to 4 mm. per hour.

The author saw the patient in consultation on June 17 and suggested the existence of a low salt syndrome. Electrolyte studies were performed immediately. Serum sodium was found to be 131.8 and potassium 5.4 mEq/L., serum chlorides 88 mEq/L. and carbon dioxide combining power 40 volumes per cent. The blood urea nitrogen was now 74 mgm. per cent. Serum proteins were 7.5 Gm. per cent, with 3.2 Gm. per cent albumin and 4.3 Gm. per cent globulin. The hematocrit was 59 per cent, with 6.00 million red cells per cubic millimeter, and 17.6 Gm. hemoglobin per 100 cc. of blood, and the sedimentation rate was 7 mm. per hour. The white blood cell counts, total and differential, were not remarkable. Urinalysis on this and on previous days showed little change from that recorded on admission, except that albumin was present only as a trace.

The electrocardiogram on this day showed a sinus tachycardia at a rate of 130 per minute, with premature contractions of auricular, nodal, and multifocal ventricular origins and a right bundle branch block. A previous electrocardiogram in 1948 had shown only prominent auricular complexes. Myocardial irritability indicated by this tracing was attributed to the intracellular myocardial effects of the low salt syndrome.

Therapy was begun June 18 with two liters of isotonic saline intravenously and replacement of drinking water with half isotonic saline, of which the patient consumed approximately three liters. Improvement was prompt, with recovery from drowsiness and improvement in weakness within two hours and the disappearance of gastrointestinal symptoms and return of appetite the next day. On this day, June 19, the electrocardiogram no longer showed the multifocal extrasystoles previously present; the right bundle branch block persisted, presumably representing right heart hypertrophy. Water and a salted diet were allowed June 19; not until June 25 was the patient returned to a low salt diet, after complete recovery and weight gain of 14.5 pounds.

Daily blood studies during the period of recovery from the low salt syndrome showed on June 18 a rise of the blood urea nitrogen to 112 mgm. per cent, while sodium, chloride, and potassium figures had reached normal (142.6, 98, and 5.0 mEq/L. respectively) and the hematocrit remained at 60 per cent. The following day the hematocrit had dropped to 55 per cent and the blood urea nitrogen to 70 mgm. per cent. The next day, June 20, the hematocrit was 48 per cent and the blood urea nitrogen 48 mgm. per cent. The hematocrit fell no further, but the blood urea nitrogen was 31 mgm. per cent on June 22 and 19 mgm. per cent on June 29.

DISCUSSION

The first case reported demonstrates that massive diuresis, to a degree greater than any of which I have been able to find record,[1] may result in no harmful effect other than the alarm of the physician. The second case indicates that the danger of electrolyte imbalance may lurk in such diuresis, as has been the reported experience of others.

An evaluation of the possible factors accounting for this distinction between the two cases is of prime interest. The rapidity and intensity of diuresis were greater in the first patient who had no electrolyte imbalance; these factors are therefore not the deciding ones. Both patients had adequate renal function, and cardiac function proved comparably restorable. While the first patient's heart failure was secondary to hypertensive and arteriosclerotic heart disease, that of the second was due to chronic pulmonary and

arteriosclerotic heart disease, but this difference is not believed to account for the observed difference in electrolyte disturbance, since that hazard is inherent in the diuresis itself. Neither patient had been restricting sodium intake prior to hospitalization for anasarca. Therapy was little different in the two cases.

Quantitative urine studies for excreted electrolytes were not carried out. Nevertheless, it is known that mercurial diuretics bring about primarily an excretion of chloride ions, secondarily of sodium ions, and only incidentally an associated water diuresis.[2] Thus, mercurial diuresis carries the hazards of hypochloremic alkalosis and, with associated hyponatremia, of the low salt syndrome.[3] On the other hand, diuresis due to digitalization of the failing heart is primarily an effect of mobilization of excess fluid, as cardiac efficiency improves.[4] The excretion of electrolytes becomes a secondary phenomenon, and, in the absence of renal damage, electrolyte disturbances do not occur.

In this distinction may lie the explanation of the fact that electrolyte disturbance was not seen in Case 1 but was present in Case 2. The former patient achieved his diuresis largely within the first six days of therapy, losing 60.5 pounds within that time, tending thereafter to a level weight. The second patient's chart shows a steady and continuous slower diuresis, approximating a straight line on the graph, over a period of three weeks. This makes it appear likely that the diuresis of the first patient was more largely a reflection of the effect of initial digitalization, whereas that of the second patient followed more closely the influence of mercurial diuresis and salt restriction. The presence of chronic pulmonary heart disease in the second patient correlates well with such a difference, in that congestive failure secondary to this cause usually responds poorly to digitalis.

SUMMARY

Massive diuresis following therapy of congestive heart failure is reported in two cases, one of which developed the low salt syndrome. An attempt is made to evaluate the factors responsible for the development of electrolyte imbalance in the one case and its absence in the other.

BIBLIOGRAPHY

1. Ramsden reported a urinary output of 481 ounces (over 30 pounds) within 24 hours after a single injection of a mercurial diuretic in a 15-year-old boy with rheumatic heart disease. (Ramsden, G. F. E.: Extreme response to a mercurial diuretic, Brit. M. J. 1:1159, 1936).

2. Schwartz, W. B., and Wallace, W. M.: Electrolyte equilibrium during mercurial diuresis, J. Clin. Invest. 30:1089-1104, 1951.

3. Zelman, S.: The low salt syndrome in congestive heart failure, J. Kansas Med. Soc. 53:219-224 (May) 1952.

4. Eichna, L. W.; Farber, S. J.; Berger, A. R.; Earle, D. P.; Rader, B.; Pellegrino, E.; Albert, R. E.; Alexander, J. D.; Taube, H., and Youngwirth, S.: The interrelationships of the cardiovascular, renal and electrolyte effects of intravenous digoxin in congestive heart failure, J. Clin. Invest. 30:1250-1261, 1951.

The Use of Hydrocortisone-Neomycin Ointment in the Treatment of Various Dermatoses

Donald L. Fuhrman, M.D., and Bruce V. Drowns, M.D.
Kansas City, Missouri

Considerable attention has recently been given to hydrocortisone acetate (Compound F) ointment in the local therapy of many dermatoses; however, little has been written regarding the use of a combination of neomycin and hydrocortisone acetate. This is a valuable addition because neomycin is an antibiotic affording a wide spectrum control of secondary infection which is commonly present.[1]

Clinical results indicate that neomycin is the local treatment of choice for cutaneous pyogenic infections except those due to Streptococcus hemolyticus, and at the same time it has a low index of sensitization. It has the added advantage of not being used parenterally so that, even if sensitization should occur, the patient has not been done a disservice in regard to future need for an antibiotic.

We wish to report our experience with the local use of a combination of hydrocortisone acetate-neomycin ointment. The preparation was a combination of these two drugs incorporated in a bland petrolatum base. Each gram contained:

Hydrocortisone acetate	10 mgm.
Neomycin sulfate	5 mgm.
Multiwax	
Cholesterol USP	
Methyl paraben	
N-Butyl-P-hydroxy benzoate	
White mineral oil	
White petrolatum	

From the Department of Dermatology, University of Kansas Medical School. Dr. H. F. Hailman of the Upjohn Company generously supplied the Neo-Cortef used in this study.

TABLE 1.

SUMMARY OF DERMATOSES TREATED WITH OINTMENT ALONE

Diagnosis	*Total*	*Cured*	*Marked Imp.*	*Imp.*	*No Imp.*
Contact dermatitis	20	10	3	7	—
Atopic eczema	12	1	5	3	3
Lichen simplex chr.	11	3	2	3	3
Seb. dermatitis	9	3	3	2	1
Pruritus ani	8	4	1	2	1
Stasis eczema	2	1	—	1	—
Nummular eczema	3	3	—	—	—
Folliculitis	1	1	—	—	—
Sun sensitivity	1	—	1	—	—
Lichen planus	1	—	—	—	1
Sycosis vulgaris	1	—	—	—	1
Psoriasis	1	—	—	—	1
Total	70	26	15	18	11

A total of 100 patients were treated and represented 19 different diagnoses. Of the 100 cases, 70 were treated with the ointment alone (Table 1). The remaining 30 cases (Table 2) received the ointment plus other forms of therapy, and we felt it not fair

TABLE 2.

SUMMARY OF DERMATOSES TREATED WITH OINTMENT PLUS OTHER FORMS OF THERAPY

Diagnosis	*Total*	*Cured*	*Marked Imp.*	*Imp.*	*No Imp.*
Contact dermatitis	9	5	1	2	1
Atopic eczema	2	1	1	—	—
Lichen simplex chr.	4	1	—	2	1
Seb. dermatitis	1	—	1	—	—
Pruritus ani	2	2	—	—	—
Stasis eczema	1	—	—	—	1
Nummular eczema	2	—	—	2	—
Moniliasis	2	1	—	—	1
Dermatitis repens	1	1	—	—	—
Pustular bacterid	3	—	—	—	3
Pustular psoriasis	2	—	—	—	2
Alopecia areata	1	—	—	—	1
Total	30	11	3	6	10

to include them in the discussion because credit could not be given to the ointment alone. Clinical evaluation of results is always difficult. We used the term "cured" to mean complete absence of signs and symptoms following therapy. "Marked improvement" indicated a dramatic immediate response and absence of signs and symptoms as long as the medication was used but some degree of recurrence when it was stopped. "Improvement" indicated that the medication was of value.

Contact dermatitis constituted our largest group and showed the best response. No attempt was made to distinguish between contact (allergic) dermatitis and external irritant dermatitis. Fifty per cent were cured while the remainder showed improvement. It was of value in all cases treated.

Atopic eczema in both infants and adults showed marked improvement in 50 per cent, while 25 per cent failed to be benefited. Lichen simplex chronicus showed excellent response in 45 per cent, but 25 per cent showed no response. Approximately 90 per cent of the cases of seborrheic dermatitis were improved. Pruritus ani showed excellent response in 62 per cent, while only 1 patient, or 12 per cent, in our series failed to show any response.

The remaining 11 cases were divided between 8 diagnoses, and it is obvious that no valid conclusions could be drawn from such a small group. Stasis eczema, nummular eczema, folliculitis, and sun sensitivity showed varying degrees of response, while lichen planus, sycosis, moniliasis, and psoriasis were not affected. It should be noted that if moniliasis is encountered, this ointment should not be used because the neomycin merely facilitates the growth of the yeast by killing off the bacteria.

COMMENTS

In all cases treated, when benefit was obtained, it was immediate and dramatic. In many instances, when the ointment was discontinued, the dermatosis recurred, and it was necessary to have the ointment in contact with the skin at all times to gain lasting benefit.

In itching dermatoses, the degree of lichenification plays a very important role; the greater it is, the less the chance of a good response. A thick heavy plaque of lichen simplex chronicus on an ankle or in the nuchal area will not show the response that a thinner lesion on the eyelid will show. On the other hand, tissues with moderate to marked swellings are sometimes helped.

We found seborrheic dermatitis to be helped greatly, as did Kile.[2] We feel the help obtained in seborrheic dermatitis was mainly in those cases where there was a marked element of secondary infection present; i.e., about the ears and in intertriginous areas. Pseudomona aeruginosa and Proteus vulgaris organisms often predominate, complicating the treatment. The response of such lesions was felt to be due in large measure to the presence of the neomycin in the ointment. We did not treat the scalp or widespread cases of uncomplicated seborrheic dermatitis.

Two patients reported some stinging sensations when the ointment was applied, and its use was discontinued. In no cases were there any reports of sensitization, and no adverse side effects were noted.

SUMMARY

One hundred cases of various skin diseases were treated topically with a 1 per cent hydrocortisone acetate ointment with neomycin added.

It was found to be of definite value in the management of contact dermatitis, atopic eczema, lichen simplex chronicus, seborrheic dermatitis, and pruritus ani. In a small group of other dermatoses, our results were inconclusive.

BIBLIOGRAPHY

1. Livengood, Clarence S., et al.: J.A.M.A. 148:334-339 (Feb. 2) 1952.
2. Kile, R. L.: Ohio State M.J. 49:1097 (Dec.) 1953.

PRESIDENT'S PAGE

DEAR DOCTOR:

As June 20 will be the last date for filing of candidates for the legislature, I wish you would give it some serious thought. This is your opportunity to select to a considerable degree the individuals who will grace our legislative chambers.

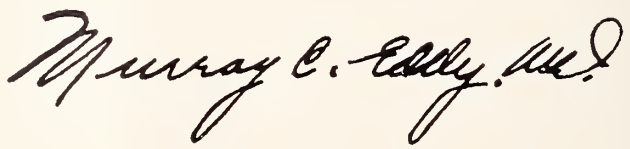
We have talked much in the past about our responsibilities of citizenship and have been proud of our democratic processes in choosing those who represent us in government. I think that now is the time to spend our efforts in selecting those who are dedicated to good government, who appreciate the freedom of the individual, and who are able and willing to make decisions.

We have been faced many times with legislatures more concerned with personal gain than the statewide problems which they must consider.

No group in the state has more friends and acquaintances than do the doctors of medicine; yet we often find, in trying to promote good health legislation, that our motives are misunderstood and our statements distorted by those who are unable to think clearly for themselves. I therefore urge you individually to devote as much time and effort as possible to helping select good candidates, and on your own local level do all that you can to help them be elected.

Medicine needs its friends in government very much to help combat the trends in socialized medicine on both state and national levels. We need our intelligent citizens in these places of great responsibility to aid in framing and passing good medical laws in our state. We need the individual with brains and integrity to serve in our legislature. Let us all do our best to help choose and elect them.

Yours sincerely,



EDITORIAL COMMENT

Chiropractors Withdraw Petitions

On approximately April 15, 1954, Dean L. Bratt and B. Wilburn Mayse, Wichita chiropractors, filed petitions in the District Court of Sedgwick County to change their names by adding "Doctor" as their first given names. Among the many attempts at evasion of the law and subterfuge that have been tried by cultists at different times, this current attempt is of interest in that it is at least singular.

In the petitions, which were identical, it was stated, "That your petitioner desires and seeks to have his name changed for the following reasons, to wit: that his present name is cumbersome, embarrassing and very difficult for strangers to understand, spell and pronounce; and that said name discredits and detracts from your petitioner's social and business standing; that an order of this Court changing your petitioner's name will be of comfort and assistance to your petitioner."

On May 6, 1954, the House of Delegates, believing this to be an abuse of a term that has regularly denoted educational attainment and is not commonly used as a given name, requested the Kansas State Board of Medical Registration and Examination to interfere in this action. This was immediately accomplished by the attorney for the board.

On about May 14, the Sedgwick County Medical Society and The Kansas Medical Society filed identical responses to both petitions. Since they are alike, only one is recorded here:

IN THE DISTRICT COURT OF SEDGWICK COUNTY, KANSAS

In the Matter of the Application of DEAN L. BRATT TO CHANGE HIS NAME	No. A-49316 Div. No. 4

RESPONSE

The Kansas Medical Society, hereinafter designated "Society," states:

1. That it is a corporation duly created and existing under the laws of Kansas and with its principal place of business located at 315 West Fourth Street, Topeka, Shawnee County, Kansas.

2. That the purposes of this Society, inter alia, are to federate and bring into one compact organization the entire medical profession of the state of Kansas; to extend medical knowledge and advance the medical science; to elevate the standard of medical education; to guard and foster the material interest of its members and to protect them against imposition and to enlighten public opinion so that the medical profession shall be more useful to the public in the prevention and cure of disease and in the prolonging and adding comfort to life.

3. That this Society has a membership of 1,824 and each and all of said members are duly licensed to practice medicine and surgery in the state of Kansas pursuant to the provisions of G.S. 1949, Chapter 65, Article 10, as amended.

4. That this Society and its members are peculiarly interested in any and all matters relating, directly or indirectly, to matters of public health and welfare in the state of Kansas and, as such, have a peculiar and direct interest in the above captioned matter and proceeding to protect the public and the members of the medical profession against imposition.

5. That the petitioner is duly licensed by the Board of Chiropractic Examiners of this state to practice chiropractic; that, as such, he may adjust by hand any displaced tissues of any kind or nature but shall not prescribe for or administer to any person any medicine or drugs now or hereafter included in Materia Medica, perform any surgery, nor practice obstetrics; that said petitioner is only authorized by law to use the title Chiropractic, D. C., and Ph.C.; that said petitioner is not learned in nor is he trained to practice any other branch of the healing art except chiropractic.

6. That the petitioner institutes and prosecutes this action under the provisions of G.S. 1949, Chapter 60, Article 23, and, as therein prescribed, there must exist a proper and reasonable cause for changing the name of the petitioner as prayed for.

7. That the petitioner desires to change his name from Dean L. Bratt to that of Doctor Dean L. Bratt and, as reasons therefor, alleges that the name, Dean L. Bratt, is cumbersome, embarrassing, very difficult for strangers to understand, spell and pronounce; that said name discredits and detracts from his social and business standing, and that an order of this court changing said petitioner's name will be of comfort and assistance to him.

8. That, under the laws of this state (G.S. 1949, 60-1005), any person shall be regarded as practicing medicine and surgery who shall use the word "Doctor", or any other title in connection with his name, which represents him as engaged in the practice of medicine and surgery; that only those who hold a license to practice medicine and surgery issued by the Board of Medical Registration and Examination of this state are entitled to the use of the word "Doctor" in connection with his name, and, therefore, the change of name sought by the petitioner herein is precluded by law.

9. That the word "Doctor" is defined as "one duly licensed to practice medicine; a surgeon"; that said petitioner, a licensed chiropractic, is specifically prohibited by law from prescribing for or administering any medicine or drugs included in Materia

Medica (that branch of science dealing with all drugs used in the treatment of diseases, their source, preparation, dosage and use) and performing surgery; that the use of the word "Doctor" by the petitioner as a part of and in connection with his name will lead the general public to believe that said petitioner is a duly licensed physician and surgeon in this state and, as such is entitled to unlimited practice rights, whereas petitioner's practice rights are expressly restricted to a single and limited branch of the healing art. That the use of the word "Doctor" in connection with the name of this petitioner would be a deception and artifice, not in good faith and truth, and contrary to law.

10. That said petitioner is attempting to accomplish indirectly that which he cannot directly or legally achieve; that he is attempting to impress the public with the thought that he is a qualified practitioner of medicine and surgery and to use a title in connection with his name permitted only by those duly licensed to practice medicine and surgery; that the allegations set forth in the petition filed herein are untrue; that no proper or reasonable cause for changing the name of petitioner, as requested, exists in fact or law; that said change of name is not sought in good faith by the petitioner but as a subterfuge to merely increase the petitioner's social and business standing and hold himself out to the general public as qualified to practice medicine and surgery in all its branches, which he is prohibited by law from doing; that the name "Dean L. Bratt" is no more cumbersome, difficult for strangers to understand, spell and pronounce, than the name "Doctor Dean L. Bratt"; that the name "Dean L. Bratt" may be more embarrassing to the petitioner than the name "Doctor Dean L. Bratt" but such does not constitute a proper and reasonable cause for changing the petitioner's name as requested; that the name "Doctor Dean L. Bratt" might increase the social and business standing of the petitioner, but such a change of name is not sought in good faith but as a subterfuge to merely increase the petitioner's social and business standing and hold himself out to the general public as a learned man and one qualified to practice medicine and surgery; that to permit the change of name, as requested, would be manifestly unfair and unjust, and misleading to the public generally, and an unlawful encroachment upon the rights and privileges of all practitioners of the healing arts and especially those practitioners lawfully licensed to practice medicine and surgery.

11. That there exists no proper and reasonable cause for the changing of the name of the petitioner as requested.

WHEREFORE, this Society requests that this court find the allegations of said petition to be untrue and that there exists no proper and reasonable cause for changing the name of the petitioner as requested; that the request of the petitioner, Dean L. Bratt, to change his name to Doctor Dean L. Bratt be denied, and that this court enter such other and further orders as may be in harmony herewith and just and lawful herein.

THE KANSAS MEDICAL SOCIETY
By Kirke W. Dale
Arkansas City, Kansas
Attorney

On approximately May 26, 1954, both petitioners requested the case to be withdrawn and, as of this time, the matter appears to have been concluded.

THE NEW COUNCIL

In the report of proceedings of the 95th annual session, recorded elsewhere in this issue, will be found an account of the revision of Council districts. These changes were voted by the House of Delegates after one year's study by the various component societies.

The change will result in a larger Council than before. Previously there were 9 officers and 12 councilors. Today 1 officer has been added and 5 additional council districts were created, whereby the present complement of the governing body of the Society consists of 27 members. While this is large, it is designed to eliminate several significant problems, and even though these are not completely solved, it appears that the situation will be improved. The proposal found apparent acceptance by the House of Delegates in that only a very few individuals objected to the changes.

In the By-Laws, Chapter VIII, the Council, Section 1, it is stated that the Council "shall have general charge of all the business affairs of this Society." It . . . "may take any action not in conflict with a former action of the House of Delegates as may be necessary to meet previously unforeseen situations, and may exercise in such cases the full power of the House of Delegates: Provided, that the Council, or the Executive Committee, may not bind this Society in any way beyond the next annual meeting of the House of Delegates."

The first consideration is that a body invested with broad powers such as given the Council should not be too small. There is, of course, the possibility of making the Council too large and thereby unwieldy. It was felt, however, that an increase of 6 persons would not create so large a Council as to interfere with its operation and would add the benefit of the additional personnel in determining policies which ultimately affect the entire Society. Therefore, the first benefit is derived from the addition of more

physicians who shall serve on the governing body.

The second advantage comes from the creation of a better distribution of representation. As previously stated, this is even now not perfect and can never be completely solved in any practical fashion. In the past, District 6 had approximately 360 members and 1 councilor representing that number of physicians. District 9 had 34 members. Each of these two councilors had 1 vote on the Council, but one of them spoke for 10 times as many doctors as did the other. In the previous 12-district Council, there were 5 districts comprised of more than 150 members each; the other 7 had fewer than 100, and several contained fewer than 50.

The present set-up has actually improved this situation only slightly, except that it has served to separate the 3 largest societies by giving a councilor to each. The spread of representation is still significant, but unless several councilors were given to Sedgwick County or the territory of some of the western districts were greatly increased, both of which plans are impractical, this problem cannot be perfectly resolved. According to the new Council, the councilor from Sedgwick County will represent 281 physicians; the councilor of District 12 will represent 24 members. There are now 7 districts containing more than 100 doctors, and there are 4 with fewer than 50.

The third consideration is that the present plan reduces the large areas that were covered in the previous districts and generally squares the boundaries where previously some were long and narrow. This should make it easier for the councilors to contact the various societies which they represent. In each district also is included at least one or more medical center. This factor, it is hoped, will provide The Kansas Medical Society with the mechanics for better interpretation of policies and will make the responsibility of the individual councilor easier to carry out.

Fourth, and finally, an improvement should be noted in that several counties which expressed a desire to affiliate with societies in other districts were changed in compliance with their wishes. There is, then, a larger Council, a somewhat improved representation and, it is hoped, a geographical improvement of the district boundaries which should create an improvement in the operating procedure of this Society.

Referrals to the Medical Center

Some weeks ago the Committee on Medical Schools, in a session held at the University of Kansas with Dean W. Clarke Wescoe and the department heads, considered the problem of patient refer-

SERVICE SEPARATIONS

As a service to physicians and communities in this state desiring additional medical personnel, the Journal of the Kansas Medical Society will publish in this column each month the names of medical officers who will shortly be separated from the armed forces. These are men who volunteered from Kansas, and many of them will probably be interested in finding locations in this state. Anyone interested in contacting these physicians may write to the address here given.

Theodore L. Batchelder, M.D.
Garden City, Kansas

Albert I. Decker, Jr., M.D.
826 Alabama Street
Lawrence, Kansas

Lyle Harris Edelblute, M.D.
1210 Thurston Street
Manhattan, Kansas

Edgar F. Ewen, M.D.
1022 East 5th
Cherryvale, Kansas

Wendell L. Good, M.D.
Perry, Kansas

Simon E. Hershorn, M.D.
Wesley Hospital
Wichita, Kansas

Everett E. Howard, M.D.
1708 North Grand
Pittsburg, Kansas

J. D. Kabler, M.D.
1506 Parker Avenue
Wichita, Kansas

J. Gilleran Kendrick, M.D.
4224 East English
Wichita, Kansas

Kenneth L. Knuth, M.D.
Atwood, Kansas

Francis X. Lenski, Jr., M.D.
703 South Cottonwood
Iola, Kansas

Stanley R. McEwen, M.D.
447 North Belmont
Wichita, Kansas

Alexander H. Milne, M.D.
2420 West 25th Street
Topeka, Kansas

Owen C. Peck, M.D.
2000 Oakland
Kansas City, Kansas

Ralph B. Reauwe, M.D.
524 West 7th
Junction City, Kansas

Julian W. Reed, M.D.
703 North Pine
Beloit, Kansas

Robert N. Shears, M.D.
15 Carlton Road
Hutchinson, Kansas

Frederick P. Wolff, Jr., M.D.
207 Edgeford Drive
Pratt, Kansas

rals to the medical center of sufficient importance to discuss with the membership of this Society. Referrals are of importance to the medical center. It is true, however, that if the referring physician understands procedures that should be followed, he may avoid delays for his patient and difficulties for the school of medicine. In the interest of providing this information, Dr. Wescoe submitted the following statement concerning the referral policies at the medical center.

"Some minor difficulties have arisen in the referral of patients to the University of Kansas Medical Center and, in order that such difficulties may be held to a minimum, the following information is provided for interested physicians.

"All referrals to the medical center should be made by a physician to a particular doctor on the medical center staff whenever that is practicable. If such is not practicable, then it is requested that referrals of patients be made by the physician to the particular department concerned at the medical center. Since it is impossible to prepare for patients at the medical center unless prior notice is given, it is requested that preliminary correspondence be carried on by the physician with the medical center in order to set up a date for appointment or hospitalization with the medical center. Because of the limitation of practice at the medical center, caused by the teaching responsibilities of the staff, it will be most helpful if patients are not sent in with a note unannounced. It is ofttimes, under those circumstances, impossible for the patients to be seen on a certain day and almost never is it possible to hospitalize such a patient because of shortage of beds."

Thanks, Jackson County

The Kansas Medical Society received a welcome word of approval recently in an editorial written by Dr. G. Wilse Robinson, Jr., for the *Weekly Bulletin* of the Jackson County Medical Society. Jackson County, being just over the state line in Missouri, is a near neighbor, and members of its medical society have been guests at a number of professional meetings in Kansas.

The editorial concerned the annual meetings of the two state medical societies, pointing out similarities and contrasts. A few comments are extracted: "Attendance is much higher in Kansas, and considering the number of members in the two states the relative attendance is very much higher. There is an air of enthusiasm there and many more scientific and technical exhibits, and the plan is entirely different, which may account for the difference in interest.

"Kansas opens its meeting with a golf tournament on Monday, and that night there is the Annual Golf Banquet. This event alone attracts a large number who otherwise would not attend . . . Once they are there they stay over a day or two for obvious reasons.

"The meeting of the Kansas House of Delegates is separate and distinct from the scientific program. . .

"The scientific program is divided into sections, there are many outstanding guest speakers as well as native talent, and all sessions are well attended. . .

"Physicians like to go to meetings to relax a bit, see old friends and learn a bit. There is some doubt as to which of the three objectives is most important."

This issue of the JOURNAL carries a report of proceedings of the 95th annual session of The Kansas Medical Society, along with reports from numerous specialty organizations and allied groups. The summary seems to bear out Dr. Robinson's view. There IS an air of enthusiasm at the Kansas meeting. Attendance records for the 1954 session show that numerous physicians from other states made it a point to be present. They were most welcome then and they'll always be welcomed in the future. We'll see you in Hutchinson in 1955!

NOTICE

The registration fee for physicians holding licenses in Kansas, payable July 1, 1954, is $3.00. Although the Kansas State Board of Medical Registration and Examination will soon send individual notices to all now registered, this special emphasis is given since the amount of the fee has been changed.

Increased administration costs and expanded activities of the Board have made necessary a change in fees. Noting that the former fee had been $1.00 per year, the 1953 session of the Kansas legislature authorized a maximum of $5.00 for this registration. Since the maximum amount will not be required in the coming year, the Board voted to establish the current fee at $3.00.

Registration Fee—$3.00
Payable July 1, 1954

95th Annual Session

The 95th annual session of The Kansas Medical Society was held in Topeka, May 2-6, 1954, with members of the Shawnee County Medical Society as hosts. Registration totals showed the following attendance: 552 members of the Society, 248 guests (out-of-state physicians, residents, technicians, etc.), 209 members of the Kansas Medical Assistants' Society, and 238 members of the Woman's Auxiliary to the Kansas Medical Society.

The program for Monday included an all-day session for members of the Kansas Chapter, American Academy of General Practice, sports events, and a tournament banquet for those who competed at golf and shooting. Scientific sessions began on Tuesday morning and continued through Thursday morning, with afternoons devoted to panel discussions. The House of Delegates held two meetings, and most specialty societies in Kansas held individual sessions. A summary of all such events is reported below.

The principal social activity was the banquet held at the Topeka Country Club on Wednesday evening, May 5, with the president of the Society, Dr. Lucien R. Pyle, Topeka, presiding. The club was filled to capacity with physicians and their wives enjoying an evening of entertainment and dancing. Dr. Pyle administered the oath of office to the new president, Dr. Murray C. Eddy, Hays, who then presented the president's key to Dr. Pyle.

AWARD TO HAROLD G. INGHAM

Mr. Harold G. Ingham, director of the Extension Program in Medicine of the University of Kansas Medical Center, was given the second Kansas Medical Society award to laymen. The award, a bronze wall plaque mounted on wood, was presented to Mr. Ingham during the banquet program. The inscription on the plaque read, "With grateful acknowledgment for outstanding services The Kansas Medical Society presents this award to Harold G. Ingham, May 5, 1954, Topeka, Kansas." The following is quoted from a letter from Mr. Ingham addressed to Dr. Pyle as president of the Society:

"I think you realize how completely taken by surprise I was, and how greatly delighted, in receiving that beautiful bronze plaque as a presentation of The Kansas Medical Society at the banquet session last week. And I regret that I am so tardy in finding an opportunity to express to you my appreciation of the spirit which prompted the granting of such an award—and of the part which you undoubtedly played in arranging this.

"I can assure you that I had not remotely suspected or anticipated a recognition of this kind. Perhaps, in my fondest dreams, I had hoped that, at the time I retired from active participation in the postgraduate medical program of The Kansas Medical Society and the K. U. School of Medicine, there might be some verbal recognition of the part I have played during the past several years in the development and promotion of the program. But I still cannot believe that the contribution which I may have made justified the action of The Kansas Medical Society in granting me the second such award ever made by that organization to a layman. The recognition is greatly appreciated and I shall forever prize the beautiful bronze plaque and the sentiment which it expresses."

FIRST SESSION, HOUSE OF DELEGATES

The first 1954 meeting of the House of Delegates was held at the Jayhawk Hotel, Topeka, on Tuesday evening, May 4. Present were 93 delegates and alternates and 24 officers, councilors, and past presidents, a total voting strength of 117. Dr. Lucien R. Pyle, president, conducted the meeting.

As usual, the time at the first meeting of the House was devoted to the presentation and approval of reports. Dr. Donald P. Trees, Wichita, chairman of the Reference Committee on Reports, summarized the reports of councilors and chairmen of standing committees as published in the April issue of the JOURNAL. All were accepted. Dr. Trees then introduced resolutions designed to enact the recommendations appearing in the reports, and all were referred to the Reference Committee on Resolutions with the understanding that action would be taken at the second meeting of the House.

Supplementary reports were given by Dr. Arthur W. Fegtly, Wichita, for the Committee on Constitution and Rules and by Dr. Barrett A. Nelson, Manhattan, for the Committee on Medical Economics. A letter from Dr. Harold H. Jones, Sr., Winfield, was read as a supplementary report for the Committee on Postgraduate Education.

Dr. Pyle reported the activities of a special committee appointed to study practice rights. A resolution on the subject, offered by Dr. L. S. Nelson, Sr., Salina, was accepted for study until the time of the second meeting of the House.

Next on the agenda was the report of the chairman of the Editorial Board of the JOURNAL, Dr. Orville R. Clark, Topeka, who was presented with a bound volume of the JOURNAL for 1953 in recognition of his service. Dr. Clark's report is published elsewhere in this issue.

Dr. Dale D. Vermillion, Goodland, constitutional

secretary, presented his annual report, stating that The Kansas Medical Society now has 1,381 paid members, 156 honorary members, 64 service members, 57 leave of absence members, and 166 delinquent members, a gain of 57 members over last year.

The report of the treasurer, Dr. John L. Lattimore, Topeka, was accepted, and the House expressed its appreciation for his services by passing with enthusiasm a motion of commendation and thanks.

Dr. Nelson, reporting for himself and Dr. John M. Porter, Concordia, as delegates to the American Medical Association, urged that all physicians who can do so attend A.M.A. sessions in San Francisco in June.

Dr. Pyle, as president, gave a brief summary of activities during his year in office and was warmly applauded by members of the House.

Dr. Murray C. Eddy, president-elect, was introduced and gave a short outline of his plans for the coming year.

During the portion of the meeting devoted to new business, three resolutions were accepted and referred to the Reference Committee on Resolutions. The resolutions were submitted by Dr. Norton L. Francis, Wichita, Dr. P. E. Hiebert, Kansas City, and Dr. R. R. Melton, Marion.

Three resolutions requiring no immediate action were accepted. The first was from the Board of Directors of the Kansas Hospital Association, suggesting that hospitals built in Kansas in the future be constructed only after a thorough study is made of available facilities in the area and the need for additional facilities. The second and third resolutions in this category, from the Kansas Pharmaceutical Association, concerned physician-pharmacist relationships.

An additional resolution from the Kansas Pharmaceutical Association, concerning a continuation of the activities of the Inter-Professional Advisory Council, was referred to the Reference Committee on Resolutions.

A letter was read from the Association of American Physicians and Surgeons, requesting co-operation of The Kansas Medical Society in conducting an essay contest. A motion that the letter be accepted and filed carried.

The president closed the meeting by announcing a schedule of events to come. He suggested that members in councilor districts 1, 2, 5, 7 and 8 hold caucuses to elect councilors before the time of the second meeting of the House.

SECOND SESSION, HOUSE OF DELEGATES

The second 1954 meeting of the House of Delegates was held at the Jayhawk Hotel, Topeka, on Thursday afternoon, May 6. Since a quorum was present, Dr. Lucien R. Pyle, president, began the meeting after a luncheon was served.

Dr. Lee H. Leger, Kansas City, chairman of the Reference Committee on Resolutions, presented the recommendations of the committee with regard to proposed amendments to the Constitution and By-Laws of The Kansas Medical Society. Each amendment, as published in the March and April issues of the JOURNAL, was voted on individually.

Approved by the House of Delegates were amendments which would achieve the following results: make the chairman of the Editorial Board a member of the Council and of the House of Delegates; provide for the nomination of three or more candidates for the offices of second vice-president and alternate delegate to the American Medical Association; make county medical societies autonomous regarding membership; require that anticipated expenses of any annual session be subject to the approval of the Executive Committee; provide that individual physicians assume responsibility for attorney fees in matters requiring the services of the Defense Board; authorize the Council to set annual dues at a figure not exceeding fifty dollars; abolish reference to fellowship classification in the American Medical Association; create a Committee on General Practice Award and outline its duties; establish a quorum for Council meetings; redistrict the state to provide for a division into 17 areas and for the election of councilors from those districts.

One proposed amendment, providing that the office of second vice-president be abolished, was defeated.

The amendments described above are printed in their entirety in another section of this issue of the JOURNAL, along with amendments approved at the 1953 meeting. Although they are officially a part of these minutes, the 1954 amendments are presented in another section to make it easier for members of the Society to make the necessary corrections in their constitutional booklets.

Dr. Leger, for the Reference Committee on Resolutions, then presented recommendations on those items of business. Passed by the House was the following:

Resolved, that the House of Delegates of The Kansas Medical Society recommend that the Committee on Allied Groups continue activity on a project whereby undertakers and ambulance drivers may be encouraged to have first aid experience and that this effort be undertaken at a county level through whatever agencies or organizations may be interested therein. The House of Delegates commends the committee for its present action.

Also approved by the House was the following:

Resolved, that the House of Delegates recommend to the professional staffs of hospitals that they con-

sider admitting doctors of dental surgery to meet with hospital professional staffs and that such individual doctors of dental surgery as appear to be qualified within their field be welcomed to perform oral surgery within the hospital.

A resolution that had been amended by the Reference Committee on Resolutions was approved by the House as follows:

Whereas, medical education must be subsidized through voluntary contributions to prevent governmental interference, and

Whereas, the physician paid only a fraction of the cost of his medical education and therefore has an obligation to the school from which he graduated and the school in this state where he earns his living, and

Whereas, in the year 1952, 3.48 per cent of all doctors and .85 per cent of Kansas doctors contributed to the American Medical Education Foundation, and in the year 1953, 8.89 per cent of all doctors and 2.42 per cent of Kansas doctors contributed, therefore be it

Resolved, that The Kansas Medical Society actively engage in raising $50,000 between this date and September 1, 1954, and be it further

Resolved, that the House of Delegates make an increased effort to raise this fund by individual voluntary contributions actively solicited through appointed representatives in each county medical society, and be it finally

Resolved, that the contributions be given to the American Medical Education Foundation but designated as gifts to the University of Kansas School of Medicine.

The following resolution was also amended by the Reference Committee on Resolutions and was passed by the House as amended:

Whereas, the danger of irrevocable loss of historical material through passage of time increases with each year, and

Whereas, the approaching centennial of The Kansas Medical Society represents an occasion during which a history would be most useful, therefore be it

Resolved, that the House of Delegates authorize the Council to appropriate a sum not to exceed $1,500 for the next fiscal year to be made available to the Committee on History, and be it further

Resolved, that each county medical society appoint a historian and that he be continued in office longer than one year and that the annual report of each county historian be sent to and recorded by The Kansas Medical Society, and be it further

Resolved, that each individual member be invited to send data on historical events of interest to The Kansas Medical Society, and be it further

Resolved, that the House of Delegates commend Dr. R. R. Melton, chairman, and his committee for their excellent planning in this regard.

Dr. Leger reported that the Reference Committee on Resolutions had carefully studied a resolution providing for educational courses for medical assistants and had approved its intent. He added, however, that the committee favored further study before action be taken. The House, at his suggestion, voted to refer the project to the Committee on Allied Groups for further study and for the planning of a curriculum.

Two resolutions presented by the Committee on Medical Schools were passed. The first reads as follows:

Whereas, at the present time the University of Kansas School of Medicine lacks qualified applicants for the student body, and

Whereas, among other reasons financial problems of students might be a contributing factor, therefore be it

Resolved, that the House of Delegates recommend that a committee be appointed to explore the establishment of a loan fund which might be used to aid qualified students who could not otherwise enter the field of medicine.

The second of the two resolutions is as follows:

Whereas, in spite of improvement which was noticeable in many directions, there is still a pronounced lack of understanding on the part of the profession regarding policies at the schools, and

Whereas, the Committee on Medical Schools of The Kansas Medical Society suggests that a continuing stream of information about the medical school should somehow reach the profession, therefore be it

Resolved, that the House of Delegates authorize the Council to accept responsibility for inaugurating such a program according to whatever means may best accomplish the desired result.

The following resolution, amended by the Reference Committee on Resolutions, was adopted by the House as amended:

Whereas, the variation in medical practice acts in the various states creates a barrier to the adoption of any immediate uniform principle, and

Whereas, no recognized accrediting agency has been recently invited to impartially examine the physical equipment nor pedagogical standards of schools in the healing profession other than those of medicine, and

Whereas, high standards of education which promise the best possible care of sick and injured cannot be maintained by the American Medical Association if the practice of medicine is diluted with inadequately trained practitioners, therefore be it

Resolved, that the House of Delegates approve that the Council of Medical Education and Hospitals of the American Medical Association may, when re-

quested by schools of branches of the healing arts that have demonstrated a belief in medical science, offer their professional services toward raising the educational standards of such schools to those of approved schools of medicine.

A resolution presented by the committee was approved by the House as follows:

Whereas, Dr. L. R. Pyle, president, Dr. M. C. Eddy, Dr. J. L. Lattimore, and Dr. George F. Gsell have rendered a significant and unselfish service to The Kansas Medical Society, therefore be it

Resolved, that the committee be commended upon its efforts, and be it further

Resolved, that the House of Delegates authorize this committee to continue in its efforts to establish the relative quality of osteopathic education as compared with the quality of medical education.

With reference to a report distributed to delegates at the first meeting of the House, on the subject of federal aid programs, the following resolution was passed:

Whereas, it appears that additional information concerning federal aid programs would be of interest to the profession, be it therefore

Resolved, that the chairman of the Editorial Board consider a series of articles upon this subject for publication in the JOURNAL.

Dr. Leger next reported on items referred to his committee at the first meeting of the House under the heading of New Business. The following resolution, introduced by the Sedgwick County Medical Society and the sixth councilor district, was approved:

Whereas, Dean L. Bratt and B. W. Mayse, chiropractors, petitioned the District Court of Sedgwick County to change their given names, alleging that their present given names are difficult to pronounce and to spell, hard to remember and embarrassing to themselves in their professional work, and

Whereas, they have each requested to change their names to "Doctor" Dean L. Bratt and "Doctor" B. W. Mayse in petitions published on the 15th of April, 1954, and

Whereas, these petitions will be heard in the District Court on or about May 14, 1954, be it therefore

Resolved, that the House of Delegates urgently request the Kansas State Board of Medical Registration and Examination to immediately consider the effects of such action on the basis that the word "Doctor" is commonly considered to be a title of professional attainment rather a proper given name, and be it further

Resolved, that this House of Delegates request the Kansas State Board of Medical Registration and Examination to immediately take any action possible to prevent a court ruling favorable to the plaintiffs.

The action of the House with reference to standard insurance forms is contained in the following resolution:

Whereas, the completion of insurance forms has become burdensome to physicians and their assistants, and

Whereas, much of the information required on forms supplied by the various insurance companies seems unimportant and in many instances a duplication of effort, and

Whereas, many of the questions cause the physician to violate his principles of medical ethics relative to divulging to a third party information that he has received from his patient, therefore be it

Resolved, that The Kansas Medical Society go on record as favoring the adoption of a uniform sickness and accident report, and be it further

Resolved, that such report forms be kept as brief as possible, and be it further

Resolved, that such reporting forms be prepared by the Committee on Medical Economics.

With reference to the Interprofessional Advisory Council the House passed the following resolution:

Whereas, the Interprofessional Advisory Council has made significant strides in its effort toward creating a better understanding among related professional groups, and

Whereas, this effort is bearing fruit through improved relationships, and

Whereas, such achievement is resulting in the improved care of the patient, therefore be it

Resolved, that The Kansas Medical Society commend Dr. C. H. Benage for his tireless effort in the establishment of this Council, and be it further

Resolved, that The Kansas Medical Society continue to support this activity.

Next considered was a resolution which made two provisions: (1) that the services of anesthesiologists, pathologists, and radiologists be referred to as "medical service" and not in terms such as "hospital service," "auxiliary service," etc., and (2) that The Kansas Medical Society oppose insurance plans directing payment for medical services rendered by a physician to a hospital. Dr. Leger's committee, reporting itself sympathetic with the resolution's intended end result, suggested that no action be taken since time did not permit detailed study and revision of the wording of the resolution so that it would accomplish its purpose. The House voted to accept the recommendation of the committee.

To conclude this portion of the meeting, the House enthusiastically voted commendation to the Reference Committee on Resolutions and to the Reference Committee on Reports for the work performed in summarizing and in making recommendations.

Dr. John M. Porter, Concordia, receiving the

necessary two-third vote of permission for the introduction of new business, made a motion that the House approve the membership of the new Council as formed by amending the Constitution and By-Laws and authorize the present councilors to retain their offices in their newly designated districts. The House, after the motion was seconded, approved.

The House also authorized the introduction of new business by Dr. Clyde W. Miller, Wichita, who presented a resolution which was approved as follows:

Whereas, The Kansas Medical Society at this time has no conclusive evidence regarding educational quality in schools of osteopathy, and

Whereas, this Society has acted to continue in its effort to obtain such information, be it therefore

Resolved, that there is no public statement to be made upon this subject at this time, and be it further

Resolved, that whatever future public statements might be made upon this subject shall have first been approved by a majority vote of the Council.

The next item on the agenda was the election of officers. The president had ballots distributed carrying the names of the candidates proposed by the Nominations Committee, and he then asked for additional nominations from the floor, presenting each office separately. There being no response from the floor, nominations were closed. Only one office, that of second-vice president, required more than one ballot, four being necessary for that election. The officers chosen are reported below.

The House then proceeded to the election of members of the Nominations Committee for 1955, results of which are reported below.

While the ballots were being counted, Dr. O. W. Davidson, Kansas City, secretary of the Kansas State Board of Medical Registration and Examination, reported that the Board had initiated action regarding the two Wichita chiropractors who have requested legal permission to change their given names to "Doctor."

Dr. Pyle expressed the appreciation of the Society to the Shawnee County Medical Society for its role as host for the 1954 meeting, and the House responded with applause. In the same way it endorsed Dr. Pyle's thanks to Dr. Dale D. Vermillion, Goodland, who had served as constitutional secretary for many years and had declined nomination for another term in that office.

At the suggestion of Dr. Barrett A. Nelson, Manhattan, the House accorded a standing vote of appreciation to Dr. Pyle for his many services to the Society.

Dr. Pyle then announced the names of the councilors for the coming year, as reported by representatives of the various councilor districts.

In closing the meeting, Dr. Pyle turned the gavel over to the new president, Dr. Murray C. Eddy, Hays, who spoke briefly on future activities before adjourning the session.

OFFICERS FOR 1954-1955

President	Dr. Murray C. Eddy, Hays
President-Elect	Dr. John M. Porter, Concordia
Immediate Past President	Dr. Lucien R. Pyle, Topeka
First Vice-President	Dr. Clyde W. Miller, Wichita
Second Vice-President	Dr. Conrad M. Barnes, Seneca
Constitutional Secretary	Dr. James A. Butin, Chanute
Treasurer	Dr. John L. Lattimore, Topeka
A.M.A. Delegate 1954-1955	Dr. George F. Gsell, Wichita
A.M.A. Delegate 1955-1956	Dr. Laurence S. Nelson, Salina
A.M.A. Alternate 1954-1955	Dr. Lee H. Leger, Kansas City
A.M.A. Alternate 1955-1956	Dr. Lucien R. Pyle, Topeka

COUNCILORS FOR 1954-1955

Printed below is a complete list of councilors, their districts, and the dates on which their terms expire:

1. Dr. Frederick E. Wrightman, Sabetha, 1957
2. Dr. Glenn R. Peters, Kansas City, 1955
3. Dr. H. Penfield Jones, Lawrence, 1957
4. Dr. Charles E. Vestle, Humboldt, 1955
5. Dr. S. A. Anderson, Clay Center, 1957
6. Dr. Floyd C. Taggart, Topeka, 1956
7. To be announced, 1956
8. Dr. James E. Hill, Arkansas City, 1957
9. Dr. L. S. Nelson, Jr., Salina, 1957
10. Dr. Harold M. Glover, Newton, 1956
11. Dr. Norton L. Francis, Wichita, 1955
12. Dr. Cyril V. Black, Pratt, 1956
13. Dr. Lloyd W. Reynolds, Hays, 1955
14. Dr. J. A. Blount, Larned, 1955
15. Dr. R. G. Klein, Dodge City, 1955
16. Dr. J. L. Jenson, Colby, 1956
17. Dr. H. Preston Palmer, Scott City, 1957

NOMINATING COMMITTEE

Five past presidents of The Kansas Medical Society were elected by the House of Delegates to name candidates for office in May 1955. Dr. Clarence H. Benage, Pittsburg, chairman of the committee, will be assisted in that office by Dr. W. F. Bernstorf, Winfield; Dr. O. W. Davidson, Kansas City; Dr. J. H. A. Peck, St. Francis, and Dr. Henry N. Tihen, Wichita.

EDITORIAL BOARD

Dr. Orville R. Clark, Topeka, chairman of the Editorial Board and editor of the JOURNAL OF THE KANSAS MEDICAL SOCIETY, was reappointed to those positions by the Council of the Society at a meeting held on May 6. At his suggestion, Dr. David E. Gray and Dr. Dwight Lawson were reappointed to three-year terms on the Editorial Board. Others

on the board are Dr. John W. Cavanaugh and Dr. Richard Greer. All are Topeka physicians.

REPORT OF JOURNAL EDITOR

During the year preceding May 1, 1954, the JOURNAL has had 852 pages, maintaining essentially the same proportion of reading material to advertising space as in the preceding year—approximately a 5:3 ratio, which is considered satisfactory for such a journal. There has been a significant increase in the amount of colored advertising, which, we believe, makes the JOURNAL more attractive.

The last 12 issues have carried 43 original scientific papers, of which 10 were received from out-of-state authors, 8 being from our 1953 state meeting. This is a significant improvement over the number of papers from state meetings as reported a year ago. There have also been 5 clinicopathological conference reports, 6 tumor conference reports, and 11 senior theses—all from the University of Kansas Medical Center. Dr. Glen R. Shepherd has again edited the CPC reports and the student theses, and the tumor conference reports have been edited by Doctors Robert E. Stowell and Chauncey G. Bly of the Department of Pathology and Oncology.

The October issue was devoted to civil defense, presenting articles on various aspects of this currently important problem. The Eighth Annual University of Kansas School of Medicine number appeared in March. Dr. Shepherd again contributed greatly to the preparation of this issue. The April issue was, as customary, devoted to the program of this 1954 session of The Kansas Medical Society. We are currently planning to present a symposium on alcoholism in an early issue.

We have now had over a year's experience with our new printer—The Ovid Bell Press, Inc., Fulton, Missouri. The entire JOURNAL staff has enjoyed this affiliation, and we feel that the service given has been of a superior quality. There has been excellent co-operation by the printer in overcoming our problems and in making suggestions for improvements in various phases of JOURNAL publication. It would seem that the Ovid Bell Press was likewise satisfied with the present arrangement as, since December 1953, we have enjoyed an unsolicited discount of 5 per cent on our total printing bill.

No attempt is being made to submit a full financial report of JOURNAL activities, but any details desired are available for any of the delegates who may wish to have them. The JOURNAL has expanded some, particularly in printed matter, and we have attempted to make it more attractive in several respects, most of which entail some additional expense. However, the income of the JOURNAL, through subscriptions and from advertising, has been, during the year just completed, $18,439.03 as against expenditures of $19,134.88, leaving a deficit of a little under $700. Though there are some regrets that the JOURNAL is not entirely self-supporting, no apology is offered for a deficit of this amount, which at this time does not make it necessary to request any additional funds from the Society.

The Editorial Board is currently composed of Doctors Dwight Lawson, David E. Gray, John W. Cavanaugh, and Richard Greer, with Doctors Glen R. Shepherd and Donald P. Trees serving as associate editors in Kansas City and Wichita. I want to take this opportunity to express my appreciation to each and every one of the members of the Board for assistance in reading and editing of papers submitted, in reading proof, and in carrying out the many details concerned with selection and preparation of material for publication. The terms of Doctors Lawson and Gray expire at this time, and it is my hope that both may be returned for another term.

I would be remiss in failing to once more thank Miss Pauline Farrell for the work which she has done so unfailingly through another year. Now in her tenth year with the JOURNAL, she has always been faithful and efficient, and has exhibited patience with the editor to a degree which defies understanding. No task concerned with the publication of the JOURNAL has been so great or so time consuming that it could discourage her, and never has she considered any to be so trivial that it received less than her full attention. It is most gratifying to have someone in her position who does actually know something about the mechanics of publication, who can keep us straight about the details we do not understand.

The services of our executive secretary, Mr. Oliver Ebel, have, as always in the past, been most valuable, and though he seldom receives due credit for his efforts, they are truly appreciated. He has done much of the editorial writing and has often furnished the much-needed insight necessary to effectively determine matters of policy for the JOURNAL. Mr. Rueben Dalbec is most welcomed in an ever-increasing role of JOURNAL production and has made significant contributions on numerous occasions. Without the fine work and co-operation of these individuals, you would have no JOURNAL, for it is truly through their efforts that the work is done.

BLUE SHIELD

Dr. Lloyd W. Reynolds, Hays, was named president of Kansas Blue Shield at the annual meeting of the Board of Directors held at the Jayhawk Hotel, Topeka, on May 2. Other officers are: Dr. Henry S. Blake, Topeka, immediate past president; Dr. Francis T. Collins, Topeka, vice-president; Dr. L. E.

Filkin, Concordia, executive vice-president, and Dr. R. P. Watterson, McPherson, secretary-treasurer.

The board is made up of the officers of Blue Shield, one physician from each of 12 geographical divisions of the state, the president and the president-elect of The Kansas Medical Society, and three lay persons. This year, Dr. Murray C. Eddy, Hays, and Dr. John M. Porter, Concordia, will represent the Society on the board.

The districts of the state and their representatives are as follows: District 1, Dr. E. T. Wulff, Atchison; District 2, Dr. P. E. Hiebert, Kansas City; District 3, Dr. F. A. Moorhead, Neodesha; District 4, Dr. E. J. Ryan, Emporia; District 5, Dr. R. P. Watterson, McPherson; District 6, Dr. James B. Fisher, Wichita; District 7, Dr. L. E. Filkin, Concordia; District 8, Dr. H. S. Dreher, Sr., Salina; District 9, Dr. Floyd L. Smith, Colby; District 10, Dr. A. M. Cherner, Hays; District 11, Dr. S. T. Coughlin, Larned, and District 12, Dr. M. F. Frederick, Hugoton.

The lay persons serving are: Mr. H. P. Reynolds, Moline, chairman of the Members' Committee; Mr. B. L. Humphreys, Hutchinson, and Mr. Martin F. Trued, Topeka.

The principal topic of discussion at the May meeting was the new non-group enrollment plan. This subject has been briefly reviewed in the Blue Shield column of former issues of the JOURNAL and will be reported on in more detail in future issues.

KANSAS ACADEMY OF GENERAL PRACTICE

The annual meeting of the Kansas Chapter of the American Academy of General Practice was held at the Jayhawk Hotel, Topeka, on May 3. The day's program included a scientific program presented by Dr. Bernard A. Watson of Clifton Springs, New York, and Dr. James R. Cook of Cleveland, a business session, and a banquet. Speaker at the banquet session was the Hon. Walter H. Judd, physician Congressman from Minnesota.

Dr. Clovis W. Bowen, Topeka, took office as president of the group. Elected to serve with him are the following: president-elect, Dr. Lawrence E. Leigh, Overland Park; vice-president, Dr. Conrad M. Barnes, Seneca; secretary-treasurer, Dr. Floyd E. Dillenbeck, El Dorado; delegate, Dr. George L. Thorpe, Wichita; alternate delegate, Dr. Clyde W. Miller, Wichita; member of board of directors, Dr. Harold L. Low, Wichita. The immediate past president, Dr. George E. Burket, Jr., Kingman, will continue as a member of the board of directors for one year, along with Dr. H. B. Sullivan, Shawnee, and Dr. Bruce Meeker, Wichita.

KANSAS STATE OBSTETRICAL SOCIETY

A dinner meeting of the Kansas State Obstetrical Society was held at the Kansan Hotel, Topeka, on May 4. Dr. Donald A. Anderson, Salina, president for 1954-1955, conducted the meeting. Dr. David E. Gray, Topeka, chairman of The Kansas Medical Society Committee on Maternal Welfare, discussed the activities of the committee with special emphasis on the study of maternal mortality. Dr. LeRoy A. Calkins, of the University of Kansas Medical Center, spoke briefly on plans for a postgraduate course in obstetrics and gynecology to be offered in the fall.

The following officers were elected to serve with Dr. Anderson during the year: president-elect, Dr. Robert L. Newman, Kansas City; vice-president, Dr. Robert M. Carr, Junction City, and secretary-treasurer, Dr. C. Donald Shrader, Newton.

KANSAS SOCIETY OF PATHOLOGISTS

The annual meeting of the Kansas Society of Pathologists was held at the Jayhawk Hotel on May 5. The following officers were elected: president, Dr. A. A. Fink, Topeka; vice-president, Dr. William J. Reals, Wichita; secretary-treasurer, Dr. Bert E. Stofer, Wichita.

KANSAS E.E.N.T. SOCIETY

Dr. James E. Hill, Arkansas City, was named president of the Kansas E.E.N.T. Society at a meeting held in Topeka at the close of the first day's scientific program during the annual meeting. Dr. D. P. Trimble, Emporia, was elected vice-president, and Dr. L. L. Calkins, Kansas City, was chosen as secretary. The delegate for 1954 is Dr. W. W. Reed, Topeka, and for 1955 Dr. William M. Scales, Hutchinson.

KANSAS RADIOLOGICAL SOCIETY

A breakfast meeting of the Kansas Radiological Society was held on May 5 at the Hotel Jayhawk, Topeka. The following officers, elected at a meeting held in Kansas City in February, are now guiding the society: president, Dr. Willis L. Beller, Topeka; vice-president, Dr. George S. Ripley, Salina; secretary-treasurer, Dr. A. M. Cherner, Hays.

KANSAS STATE PEDIATRIC SOCIETY

The newly organized Kansas State Pediatric Society met at the Topeka Country Club the evening of May 4. Plans were made for a one-day postgraduate course in pediatrics to be offered Kansas physicians early in the fall, probably in September. Details of the plan will be announced later.

A meeting of the Kansas Chapter, American Acad-

emy of Pediatrics, followed. Dr. B. I. Krehbiel, Topeka, was elected state chairman.

KANSAS ORTHOPAEDIC CLUB

A meeting of the Kansas Orthopaedic Club was held at the Hotel Jayhawk, Topeka, on May 5. Dr. C. K. Wier, Wichita, was elected president of the group, and Dr. H. O. Marsh, Wichita, was re-elected secretary-treasurer.

A scientific program was given by a group of Topeka physicians, Dr. Joseph Gendel, Dr. Clyde B. Trees, Dr. G. B. Joyce, and Dr. M. E. Pusitz. A social hour followed the meeting.

KANSAS PSYCHIATRIC SOCIETY

The Kansas Psychiatric Society held its annual meeting at Lawrence in April. Dr. R. C. Anderson, Topeka, took office as president. He will be assisted by the following: Dr. I. Clark Case, Topeka, president-elect; Dr. Arthur F. Dundon, Topeka, secretary-treasurer, and Dr. C. J. Kurth of Wichita and Dr. William Rottersman of Topeka, councilors.

KANSAS HEART ASSOCIATION

Dr. Clarence W. Erickson, Pittsburg, took office as president of the Kansas Heart Association at the close of the organization's annual meeting held at the Lassen Hotel, Wichita, May 21. Other officers chosen are: president-elect, Dr. G. Loren Norris, Winfield; vice-president, Mrs. John Nelson, Topeka; secretary, Mr. Frank Sullivan, Topeka; treasurer, Mr. Willard Briedenthal, Kansas City.

Dr. Lee H. Leger, retiring president, was speaker at the dinner session, having as his subject "Rehabilitation of the Cardiac." The executive director of the American Heart Association, Mr. Rome A. Betts, spoke at the luncheon meeting. The scientific portion of the program consisted of a panel discussion on rheumatic fever by Dr. Earl L. Mills, Dr. William F. McGuire, Dr. Howard Wagenblast, and Dr. Alfred M. Tocker, all of Wichita. Dr. Erickson served as moderator.

KANSAS SOCIETY OF ANESTHESIOLOGISTS

The annual meeting of the Kansas Society of Anesthesiologists was held at the Hotel Jayhawk, Topeka, on May 4. The following officers were elected: president, Dr. E. Wray Enders, Kansas City; vice-president, Dr. Walter Stephenson, Norton; secretary, Dr. Dale U. Loyd, Wichita; treasurer, Dr. Lila J. Gairns, Topeka.

The principal discussion concerned the establishment of an anesthesia study commission, but no action was taken. The society expressed its satisfaction with Blue Shield policies on anesthesiology.

COLLEGE OF CHEST PHYSICIANS

The annual meeting of the Kansas Chapter, American College of Chest Physicians, was held at the Kansan Hotel, Topeka, on May 4. Dr. A. L. Ashmore, Wichita, was elected president of the group, Dr. C. J. W. Wilen, Manhattan, was named vice-president, and Dr. Martin J. FitzPatrick, of the University of Kansas Medical Center, was chosen as secretary. A scientific program, arranged by Dr. Andre Baude, Topeka, was presented. Dr. C. F. Taylor, Norton, is governor of the college for Kansas.

SPORTS EVENTS

Sports minded members of The Kansas Medical Society enjoyed golf and shooting on the first day of the 1954 annual meeting with a tournament banquet at the Topeka Country Club in the evening.

For the second straight year, the golf championship was determined by a flip of a coin, and for the second straight year Dr. H. Penfield Jones, Lawrence, lost. This year Dr. Robert P. Norris, Wichita, was winner. Each scored an 80. Dr. Norris received the championship trophy given by Quinton-Duffens Optical Company.

Other winners in the championship flight were Dr. Fred N. Bosilevac, Kansas City, third low gross; Dr. A. W. Bradford, Overland Park, fourth low gross; Dr. H. Lee Barry, Wichita, fifth low gross; Dr. Fred L. Ford, Topeka, low net; Dr. W. L. Pratt, Leavenworth, second low net; Dr. J. Robert Weaver, Wichita, third low net.

The following were winners in the first flight: Dr. M. C. Rucker, Sabetha, first low gross; Dr. J. F. Nienstedt, Beloit, second; Dr. John L. Lattimore, Topeka, third; Dr. Hubert M. Floersch, Kansas City, fourth; Dr. Wilson K. Hobart, Topeka, fifth; Dr. Weir Pierson, McPherson, low net; Dr. A. E. Hiebert, Wichita, second; Dr. T. L. Hill, Arkansas City, third; Dr. M. W. Carlson, Ellinwood, fourth; Dr. Byron J. Ashley, Topeka, fifth.

In the second flight Dr. D. Paul Trimble, Emporia, had low gross; Dr. J. H. Holt, Wichita, second; Dr. Fred J. McEwen, Wichita, third; Dr. Floyd L. Smith, Colby, fourth; Dr. George D. Marshall, Colby, low net; Dr. E. W. Christmann, Manhattan, second; Dr. L. C. Joslin, Harper, third; Dr. Leo K. Crumpacker, Wichita, fourth.

Dr. G. E. Milbank, Wichita, had low gross in the third flight. Other winners were: Dr. Orville R. Clark, Topeka, second low gross; Dr. Mac F. Frederick, Hugoton, third; Dr. Ray A. West, Wichita, fourth; Dr. Victor H. Hildyard, Baldwin, low net; Dr. L. G. Schwartz, Topeka, second; Dr. Alex C. Mitchell, Lawrence, third; Dr. R. A. Crawford, Hutchinson, fourth.

Trapshooting prizes were won by Dr. W. A. Smi-

ley, Sr., Junction City, first; Dr. George L. Gill, Sterling, second; Dr. Arnold H. Baum, Dodge City, third; Dr. Shirley E. Clark, Topeka, fourth; Dr. R. E. Cheney, Salina, fifth; Dr. F. L. Loveland, Topeka, sixth; Dr. Dale D. Vermillion, Goodland, seventh; Dr. Murray C. Eddy, Hays, eighth; Dr. C. L. Scuka, Wichita, and Dr. W. A. Smiley, Jr., Junction City, ninth; Dr. R. Dale Dickson, Topeka, tenth; Dr. Howard U. Kennedy, Topeka, tyro.

In addition to the golf championship trophy awarded by Quinton-Duffens Optical Company, the following prizes were given: Endee bag, A. S. Aloe Company; Ray-Ban goggles, Bausch and Lomb Optical Company; sterling silver cuff links, Armour Laboratories; Kodak Duaflex, White Laboratories, Inc.; gift set, John H. Breck, Inc.; Ronson lighters, Ciba Pharmaceutical Products, Inc.; desk set, U. S. Vitamin Corporation; golf balls, William S. Merrell Company; onyx homestead lighters, Winthrop-Stearns, Inc.; golf balls, Eli Lilly and Company; electric clock, Abbott Laboratories; fifth of Scotch, E. R. Squibb and Sons; golf balls, Doho Chemical Corporation; G. E. dark adaptor fluoroscopic goggles, General Electric Company; gift packages of baby foods, Gerber's Baby Foods; sportsman's set, Harrower Laboratories, Inc.; gold golf tees, Hoffman-LaRoche, Inc.; golf balls, Mead Johnson and Company; Endee bags, Munns Medical Supply Company, Inc.; golf balls, Ortho Pharmaceutical Company; pint of vanilla extract, Parke, Davis and Company; golf balls, Charles Pfizer and Company, Inc.; book *Current Therapy,* W. B. Saunders Company; ashtrays, Smith, Kline and French Laboratories; book *The Gold-Headed Cane,* Charles C Thomas, Publisher; desk thermos bottle, Roerig and Company; wallet, Lincoln Laboratories.

Newly elected officers of the Kansas Medical Golf, Trap, and Skeet Association, chosen at a business session following the banquet, are as follows: president, Dr. Fred L. Ford, Topeka; vice-president for golf, Dr. David Taylor, Norton; vice-president for trap, Dr. C. L. Scuka, Wichita; secretary-treasurer, Dr. Fred N. Bosilevac, Kansas City; directors, Dr. Murray C. Eddy, Hays; Dr. William A. Smiley, Sr., Junction City; Dr. John L. Lattimore, Topeka; Dr. R. Dale Dickson, Topeka; Dr. James W. Shaw, Wichita, and Dr. Hubert M. Floersch, Kansas City; publicity director, Dr. R. A. Crawford, Hutchinson.

WOMAN'S AUXILIARY TO K.M.S.

Mrs. E. R. Millis, Kansas City, was installed as president of the Woman's Auxiliary to The Kansas Medical Society at the close of the group's annual meeting held in Topeka, May 3-6. Mrs. Barrett A. Nelson, Manhattan, was chosen as president-elect; Mrs. James L. Jenson, Colby, first vice-president; Mrs. Herbert R. Schmidt, Newton, second vice-president; Mrs. Wayne O. Wallace, Atchison, third vice-president; Mrs. E. M. Harms, Wichita, recording secretary; Mrs. Louis G. Graves, St. John, treasurer, and Mrs. Lee H. Leger, Kansas City, corresponding secretary.

MEDICAL CLINIC MANAGERS

The annual meeting of the Kansas Association of Clinic Managers was held at the Kansan Hotel, Topeka, May 4. Mr. Arthur Holtman, Manhattan, was elected chairman; Mr. Robert Jay, Arkansas City, vice-chairman, and Mrs. Madeline Deal, Liberal, secretary-treasurer.

KANSAS MEDICAL ASSISTANTS' SOCIETY

The 14th annual meeting of the Kansas Medical Assistants' Society was held at the Hotel Kansan, Topeka, on May 2 and 3, with 209 members in attendance. Miss Agnes Burns, Kansas City, was installed as president for the coming year at the close of the meeting, and the following additional officers were elected: president-elect, Mrs. Pauline Keller, Topeka; vice-president, Miss Mary Ellen Babb, Wichita; secretary, Mrs. Ruth T. Groves, Mission, and treasurer, Mrs. Louise Buckley, Emporia. Mrs. Carmen Kline, Kansas City, was named as corresponding secretary.

Miss Bessie Parker, Emporia, was chosen councilor of District 2 to represent members in that section of the state, and Mrs. Irene Weathers, El Dorado, was elected councilor of District 3. Holding over until 1955 are Miss Pauline Farrell, Topeka, District 1; Mrs. Hazel Fletcher, Hays, District 4, and Miss Carmen Jiminez, Syracuse, District 5.

Board of Health Appointments

Two physicians, Dr. H. St. Clair O'Donnell of Ellsworth and Dr. H. Penfield Jones of Lawrence, were reappointed to three-year terms on the Kansas State Board of Health last month. Other physicians serving on the board are Dr. Dick B. McKee, Pittsburg, whose term expires in 1955; Dr. E. A. McClintock, Topeka, 1955, and Dr. Robert C. Polson, Great Bend, 1956. Also serving on the board are a dentist, a pharmacist, a veterinarian, a sanitary engineer, and a hospital administrator.

Cancer Grant to K.U.

A grant of $6,243 from the American Cancer Society for research on abdominal tumors has been awarded Dr. J. K. Frenkel, pathologist at the University of Kansas Medical Center.

Hemangiomas and Lymphangiomas

Tumor Conference

Edited by C. G. Bly, M.D., and Irwin Joffe, M.D.

Dr. Bly: Today we will be primarily concerned with benign tumors of small blood and lymph vessels. Willis entertainingly summarizes the dissenting view that most of these "angiomas" are not really tumors, since they are so often congenital and have very limited if any growth potential beyond that of the host.[1] We have one patient with a lymphangioma and three with hemangiomas.

Cancer teaching activities at the University of Kansas Medical Center are aided by grants from the National Cancer Institute and the Kansas Division of the American Cancer Society. Dr. Bly is a Scholar in Cancer Research of the American Cancer Society. Dr. Joffe is a Trainee of the National Cancer Institute.



Figure 1. (Case 53-86). (a) Swelling of neck due to cystic hygroma; (b) large cystic (collapsed) cell-free lymphatic spaces, smooth muscle cells in center of field, and aggregated lymphocytes in lower right field (x42).

Dr. Ye: The first patient (Case No. 53-86) is a 13-year-old white boy who was admitted to the University of Kansas Medical Center for surgical removal of a swelling on the left side of the neck. This swelling had been first noted six months before admission and had gradually increased in size. Repeated aspirations of the mass had been done, with temporary decrease in size on each occasion. There had been no limitation of motion of the neck, pain, or dysphagia.

On physical examination the large cystic swelling, about 4 cm. in diameter, was seen in the left side of the neck anterior to the sternocleidomastoid muscle (Figure 1a). The swelling seemed to contain fluid but did not transilluminate; it was not fixed to the overlying skin. The trachea was in the midline, and there was no palpable enlargement of the thyroid or cervical lymph nodes. Two days after admission, the mass was excised under general anesthesia. The boy returned home on the first postoperative day.

Dr. Boley: This is a lymphangioma or cystic hygroma. When the gross specimen was incised in the pathology laboratory, it collapsed gradually as clear fluid was released from the multiple thin-walled partially or completely septated compartments. Occasionally blood-tinged fluid was noted, possibly related to operative trauma. On the microscopic slide, we see (Figure 1b) large and small collapsed cell-free cystic spaces, lined by flat endothelial cells. Focal and diffuse lymphocytes seem almost to be integral parts of these tumors. Dilated lymphatic vessels, possibly part of the tumor, appear to enter a small and otherwise normal cervical or supraclavicular lymph node adjacent to the tumor tissue.

Dr. Bly: Dr. Robinson, would you give us some ideas as to the differential diagnosis, characteristics, and treatment of such a tumor as this one.

Dr. Robinson: One might briefly consider involvement of the lymph nodes by various lymphomas or tuberculosis, deep hemangioma, neurofibroma, lipoma, branchial cyst, or even a laterally displaced thyroglossal cyst.[2, 3] However, from the facts that this tumor was large, unilateral, flaccid, and had been repeatedly aspirated, we felt sure it was a typical cystic hygroma. The cystic hygroma is a lymphangioma of the neck and contains many locular spaces of variable size, each containing straw-colored fluid, clear or slightly turbid. Transillumination may be difficult if the fluid is cloudy or hemorrhagic,

if the walls of the spaces are thick, or if the spaces are small.

These are not uncommon tumors—we see 3 to 5 each year. They occur most commonly in children. They are most frequently found in the soft tissue of one side of the neck. However, lymphangiomas also occur with decreasing frequency in such lymphoid areas as the axillary, inguinal, and mediastinal regions,[3] tongue, face, lips,[2] viscera,[1] or anywhere in the body.

The best treatment of this tumor is surgical excision. The entire tumor can be safely and easily removed if it is small, superficial, or well encapsulated. However, some tumors may have so deeply infiltrated or surrounded local tissues that total removal might entail damaging the contiguous structures beyond possible repair. In such a case, the surgeon may resect that part of the tumor that can be safely excised and leave behind the inaccessible remainder. Small remnants or fragments of tumor left behind at operation will not normally result in recurrent growths. On the other hand, some recurrences have been said to occur even after radical neck dissection.[2]

These growths can at times be extensive. One of the most surgically formidable cystic hygromas I have ever encountered occurred in a tiny infant. The tumor mass extended from the base of the skull down to and behind the clavicle and into the superior mediastinum; it extended deeply, from the skin posterior to the sternocleidomastoid muscle, and medially, to surround the esophagus, larynx, pharynx, and carotid sheath. All of the important anatomical structures were identified, dissected clean, and salvaged, with resection of almost all of the tumor.

Acute emergencies and more chronic complications must be considered in addition to the cosmetic reasons for removing these often unsightly tumors. We have been forced to operate upon some little children at inopportune times because obstruction of the airway was present or impending. In this situation, a tracheotomy is performed; the tumor is removed later. The acute obstructions may be caused by mechanical pressure from (a) the spontaneous hemorrhage which occurs into the locules of the tumor, or (b) the inflammatory edema, which may accompany the above hemorrhage or which may involve adjacent lymph drainage areas and lymph nodes, and sometimes even the tumor tissue itself, during upper respiratory infections. The chronic obstructions may be caused by (a) contraction of the fibrous scars resulting from the healing of severe inflammatory lesions, or (b) the slow growth or expansion of the tumor itself. Other complications include infection of the lymphangioma and malignant transformation, the latter being conceivable but not yet seen by me.

Dr. Joffe: Would you comment on the therapeutic uses of aspiration of fluid from these lesions, or of injections of sclerosing solutions, and on the expected mortality in the surgical treatment of these lesions?

Dr. Robinson: Aspiration of fluid is helpful in the diagnosis and in the temporary palliation of these tumors. It is not curative, and one must keep in mind the chances of hemorrhage and infection if it is improperly done. Sclerosing solutions have been injected, but with poor success, in some attempts to shrink large and difficultly operable lymphangiomas. In the hands of a competent experienced surgeon, the operative mortality is low—probably lower than that of giving a general anesthetic.

Dr. Bly: Dr. Boley, do you regard these tumors as benign neoplastic growths or as lymphangiectasia or lymphatic varicosities?

Dr. Boley: The lymphangiomas in some areas may be regarded as hamartomas: congenital accumulations in abnormal proportions and arrangements of tissue elements which are normally found in those situations and which usually grow only as long as the host grows. They might arise as a result of some localized congenital diathesis to lymphangiectasia, except that they do not seem to be connected with, or at least able to drain cumulated fluid into, the normal lymphatic channels. Goetsch believed that these were true neoplasms composed of proliferating endothelial buds.[4] However, many pathologists now agree that these expansions of the tumor tissue represent merely passive insinuation of endothelialized spaces into adjacent tissue, possibly in response to increasing hydrostatic pressure.

Dr. Tice: Lymphangiomas are not considered to be problems for irradiation therapy, except at dosages that would damage nearby tissues. Radon seeds and x-rays have been tried with mixed success in cases with mediastinal remnants.[3]

Dr. Bly: Since the next cases are all concerned with hemangiomas of various types, it might be well to hear first all of the case histories and then move to broader discussions.

Dr. Ye: The first of these cases (Case 53-87) is an 11-month-old white girl who was admitted with a bright red elevated soft lesion on the left labium majus (Figure 2a). This lesion had gradually enlarged in diameter from about ½ cm. at birth to about 3 x 2 cm. at admission. This was considered to be a typical cavernous hemangioma and was removed by local excision. No complications were encountered.

The next case (Case 53-88) is a 4-year-old white boy who was admitted to the hospital for surgical treatment of a large diffuse tumor deep within the substance of the upper lip. It had been present since

birth and was considered to be a hemangioma. It had been treated first by injections of sclerosing solutions, later by x-ray therapy in this hospital. Although the tumor had decreased in size with treatment, the upper lip remained larger than normal (Figure 3a). Under general anesthesia, the greater part of the hemangioma was excised and plastic revision of the lip was performed.

The last case (Case 53-89) is a 7-month-old white boy bearing on his scalp a 3 x 3 cm. flat red hemangioma which had been present since birth. It had enlarged rapidly during the first few months of life but remained stationary in size during the last month. Excision of the hemangioma was performed without complications, and he was dismissed three days after admission.

Dr. Boley: If tiny capillary-sized blood spaces predominate, one calls it a capillary hemangioma; similarly, if larger venule-sized spaces with thicker walls predominate, one calls it a cavernous hemangioma. Most hemangiomas, however, contain mixtures of capillaries, venules, and even small arteries, in varying proportions.

The specimen from the vulva (53-87) consisted of a 3.5 x 3 x 2 cm. ellipse of mottled blue skin and subcutaneous tissue, containing grossly visible vascular spaces. The microscopic slide shows areas of thin-walled small capillary channels (Figure 2b), larger thicker-walled venules or cavernous channels, and occasional very thick-walled small arteries, all lined by endothelial cells and containing erythrocytes. Although most of the hemangioma is of the capillary type, including a few very cellular and poorly canalized fields, there are scattered large and small cavernous (venule) areas to account for the raised character and bluish coloration of the gross lesion.

The specimen from the lip (53-88) measured 2 x 1.5 x 1 cm. It appeared grossly to be vascular fibro-adipose tissue with an epithelialized surface. In the microscope slide, one finds rare scattered thin-walled, and few thick fibrous-walled, dilated blood vessel spaces (Figure 3b). Dense fibrous tissue bands, probably resulting from sclerosing and irradiation

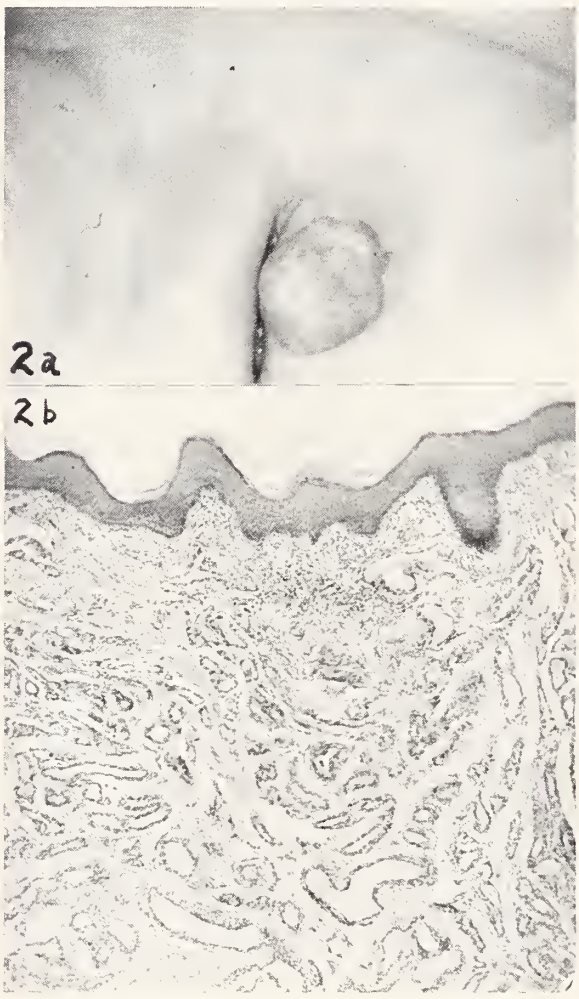


Figure 2. (Case 53-87). (a) Cavernous hemangioma of vulva; (b) mixture of numerous dermal capillary and cavernous (venule) blood spaces (x42).

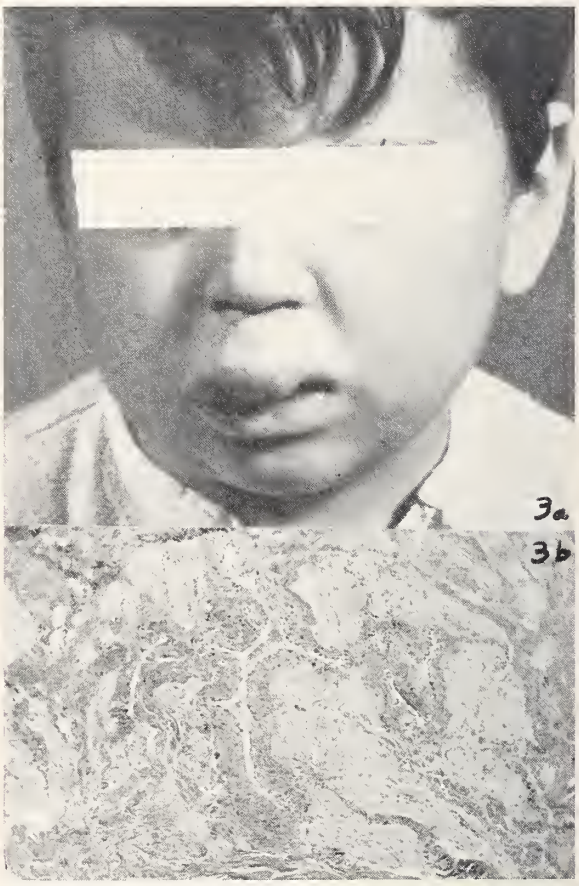


Figure 3. (Case 53-88). (a) Upper lip, still enlarged after various treatments; (b) large cavernous blood spaces, with greatly thickened walls blending into dense fibrous scar tissue between muscle bundles (x42).

therapy, extend through the skeletal muscle and surround or partially obliterate remaining cavernous blood spaces. One cannot tell now whether this hemangioma had any large capillary component before treatment.

The elliptical specimen of skin from the scalp of the last patient (53-89) contained a central 1 cm. vascular reddish-blue soft area. The microscopic slide shows predominantly capillary blood spaces with occasional cavernous spaces.

Dr. Robinson: Although the advantages of anatomical classifications are well understood, a strict morphological classification of hemangiomas into capillary or cavernous types is inadequate. In evaluating these tumors, their location, physical appearance, and response to treatment must also be considered. In general, it can be said that these lesions vary from small spider hemangiomata or foci of dilated capillary channels just under the skin, to great locular collections of blood which are found in some of the cavernous type of hemangiomas. Between these limits we see all admixtures of morphologic types. They may be found in any area of the body, but most of them which are detected are subcutaneous or dermal, most are present at birth, and most are distributed over the head and neck. The questions of congenital versus acquired origin, neoplasia versus simple expansion, are similar to those above for lymphangiomas. The tumors may become hemorrhagic, ulcerated, infected, or thrombosed. In contrast to the uncertain connections of lymphangiomas, most hemangiomas are said to possess direct connections with the circulating blood stream, through afferent and efferent blood vessels.

Clinically, hemangiomas are classified into three main groups.

A. The first group, the capillary hemangiomas, is typified by the so-called "port-wine stain." It is made up of superficial capillary dilatations, relatively mature endothelium, under and within the skin. This type of lesion is not elevated. It is radio-resistant and does not spontaneously regress. The texture of the affected skin is quite the same as that of the surrounding skin although of different color. This type of tumor, in our opinion, should not be treated by any means, since therapy may have unpredictably disastrous results. We usually recommend the use of cosmetics. Girls don't have the problem of social stigma which relates to the use of cosmetics by boys.

Excision of the lesion, followed by skin grafting, has been tried. A major cosmetic difficulty of this method is the pigment deposition which frequently occurs in the graft. The graft then has the obvious appearance of a patch even though it may have normal texture. If the pigmentation of the graft requires the use of cosmetics, nothing has been gained by the operative treatment of "port-wine stains." I think almost everyone now agrees that this type of lesion should not be treated by irradiation. The ultimate response of the tumor is usually poor, and if very much treatment is given, radiodermatitis and conceivably carcinoma may develop many years later. Injections of sclerosing materials and electro-coagulation are practically valueless. Tattooing has had its proponents, but since one can introduce only a pigment of one fixed color, the tattoo cannot vary with normal seasonal changes in skin color, and cosmetics must still be used. Dry ice has been widely used to produce superficial destruction of the outer layers of the skin and subsequent scarring of the underlying dilated capillary bed, but it can no longer be recommended as treatment for this type of lesion.

B. The second group of hemangiomas recognized clinically is typified by the so-called "strawberry mark." This lesion is also primarily a capillary hemangioma, but is a much more cellular tumor than the "port-wine stain." Clinically it is a slightly raised lesion. It often begins in infants as a tiny lesion, subsequently grows rapidly and may spread to involve many neighboring structures, then tends to stop growing and may eventually completely regress. The "strawberry" hemangiomas respond best to irradiation therapy and, since not all regress, should be treated with irradiation as early in life as possible. Excision of this type of angioma often does unnecessary damage to deeper contiguous anatomical parts, particularly if the lips or eyelids are involved. Injection of sclerosing solutions and application of dry ice are, in my opinion, of no value for this type of lesion.

C. The third general group of these tumors includes the cavernous hemangiomas. These are made up of multiple interconnected locules, often with thick walls, and have a mature type of endothelial lining. The cavernous hemangioma often contains considerable amounts of blood, which can be emptied by manual pressure. When the examiner removes his hand, the tumor slowly fills again. If the patient strains or cries, particularly if the angioma is located on the head or neck, the tumor may fill up rapidly and turn a dusky purple color. This type of lesion needs definitive treatment because it tends to spread, or to become ulcerated and infected, producing soft tissue destruction. It neither grows nor spreads as rapidly as does the "strawberry" angioma, nor does it spontaneously regress.

Surgery is certainly indicated in these lesions which are discrete and can be removed without much local damage, such as those in the loose skin of the scalp, back, and forehead. Another technique involves coagulating the blood within locular spaces of cavernous hemangiomas by using an electric needle.[5] As the coagulated blood becomes organized and the fibrous scar contracts, the tumor shrinks. This

procedure works well in some instances, but at times it may be followed by ulceration and deep-seated infection. Injection of sclerosing agents into the cavernous hemangioma also has some therapeutic value. The introduction of the irritative substance, such as morrhuate, damages the endothelium. This results in thrombosis, organization, and shrinkage of the tumor just as after freezing, electro-coagulation, or irradiation.

If these tumors are not discrete, or if they involve structures such as the eyelid, inner canthus, clitoris, or posterior throat, which are often badly damaged by surgery or other means of therapy, irradiation may be the only acceptable treatment.

In an occasional case, the types of hemangioma tissue are so mixed that the tumor defies both adequate classification and treatment.

Dr. Tice: I would agree with Dr. Robinson's clinical classification. Although it is common for "strawberry" hemangiomas to disappear spontaneously, it is also common for them to grow larger, become ulcerated, and cause soft tissue destruction with scars. Too often the patients or parents are advised by the doctor to wait and see what happens, and during this interval the lesion increases in size and ulcerates. Moreover, the radiosensitivity of hemangiomas decreases markedly after the first two years of life, so one cannot simply wait for regression.[6]

We treat the flat "strawberry" hemangioma with a 10 mg. radium plaque. A dose calculated to be a little less than an erythema dose is applied. The child is then seen again in two months. If there has been definite fading of the lesion, a treatment is not given again. The child is watched over the next year, and as long as there is definite regression of the lesion and fading, treatment is not given. If the regression stops, we do not hesitate to give a second or a third treatment over a period of two or three years. Plaques containing radioactive phosphorus have also been used.

In the group of cavernous hemangiomas, we have had excellent results with deep therapy. We use 200 kv. x-ray, relatively heavy filtration, and cross fire the lesion through multiple small ports, without applying an erythema dose to any one area. One of our most successful cases of this type was a baby girl who had a large hemangioma of the breast. Radiation, using this procedure on two different occasions six months apart, resulted in satisfactory regression of the hemangioma.

In the treatment of hemangiomas with irradiation, one must always keep in mind the possibility of damaging intervening, nearby, or underlying normal tissues. Undue repetition of an initially safe dosage may lead to necrosis of the skin, radiodermatitis, devitalization of the skin, or, on rare occasion, malignant change. This is true whether one uses plaques of radium or P^{32}, radon seed, or radiation instruments with varying voltage levels. Treatment about the face requires consideration of possible damage to the erupting teeth. In a child, four years old, who had a diffuse cavernous hemangioma of the soft tissues of the forearm, great care was taken to protect the epiphyseal centers. Even with this protection, he now, 10 years later, has about 2 cm. of shortening of the treated arm compared to the other arm. Whether the treatment is surgery or radiation, one must always keep in mind the fact that when we get through the patient must be better than before the treatment was started, or the treatment has not been adequate. In one series of 225 cases with cavernous hemangiomas, treated by radiation, fully satisfactory therapeutic results were obtained in over 80 per cent of the cases, including many which had been followed for more than 10 years.[6]

BIBLIOGRAPHY

1. Willis, R.: Pathology of tumors, pages 700-715, Butterworth, London, 1953.
2. Maxwell, J. H.: Tumors of the face and neck in infancy and childhood, Southern Med. J. 45:292-299, 1952.
3. Gross, R. E., and Hurwitt, E. S.: Cervicomediastinal and mediastinal cystic hygromas, Surg. Gyn. and Obstr. 87:599-610, 1948.
4. Goetsch, E.: Hygroma colli cysticum and hygroma axillare, Arch. Surg. 36:394, 1938.
5. Figi, F. A.: Hemangiomas of the mouth, Ann. Otol. Rhin. Laryng. 56:853, 1947.
6. Thoms, J., and Fjeldborg, N.: Radiation treatment of hemangiomas, Acta Radiol. 40:39-53, 1953.

COUNTY SOCIETIES

A meeting of the Doniphan County Society was held at Denton late in April. Dr. Emerson D. Yoder, Denton, was elected president, and Dr. F. E. Totten, Wathena, was named secretary-treasurer.

Dr. George F. Gsell, Wichita, was guest speaker at the April meeting of the Cowley County Society held at Winfield. His subject was "Functions and Diseases of the Eye."

A meeting of the Wyandotte County Society was held at the City-County Health Building, Kansas City, on April 20. Dr. LeGrand B. Byington spoke on "The Program of the Health Department," and Dr. Calvert Winter discussed "Communicable Diseases."

A new project of the Wyandotte County Society is the establishment of an Emergency Medical Service which will operate 24 hours a day. Advertising space was purchased in the *Kansas City Kansan* to acquaint residents of the community with the services available.

A clinicopathological conference featured a meeting of the Sedgwick County Society held at the

Lassen Hotel, Wichita, on May 11. Taking part were Dr. William J. Reals, Dr. William P. Callahan, Jr., and Dr. Bert E. Stofer.

The scientific program of the Wyandotte County Society, at a meeting held in Kansas City on May 18, included two papers, "Early Recognition of Speech Defects," by R. L. Schiefelbusch, Ph.D., and "Management of Head Injury," by Jack R. Cooper, M.D. At a business session which followed, Dr. Glenn R. Peters was named councilor for the new district compromising Wyandotte County.

ANNOUNCEMENTS

Kansas physicians who desire reprints of a paper presented by Dr. Bernard A. Watson, Clifton Springs, New York, on the subject of the hypometabolic state are asked to send their requests to the JOURNAL. Although other papers by Dr. Watson will be published in the JOURNAL later, this one had been promised for publication elsewhere.

The Department of Otolaryngology, University of Illinois College of Medicine, announces its basic science course in otolaryngology offered by its affiliated hospitals. This combined postgraduate course and residency will begin its 1954-1955 session on July 1, 1954, with other openings later. Stipends range from $1,320 annually to $1,920. Application forms are available from the department, 1853 West Polk Street, Chicago 12.

The Eighth Annual Rocky Mountain Cancer Conference will be held in Denver, July 14-15, at the Shirley-Savoy Hotel. There is no registration fee for the conference, sponsored jointly by the Colorado State Medical Society and the Colorado Division of the American Cancer Society. The program will include eight guest speakers covering radiology, dermatology, surgery, urology, pathology, internal medicine, and gynecology.

A refresher course for general practitioners on the subject of common skin conditions will be offered in Denver, July 16-17, under the joint sponsorship of the University of Colorado School of Medicine and Fitzsimons Army Hospital. Applications should be sent to Director of Graduate and Postgraduate Medical Education, University of Colorado School of Medicine, 4200 East Ninth Avenue, Denver. A registration fee of $5.00 must accompany each application, and a tuition fee of $20 will be charged.

A course in "Newer Developments in Cardiovascular Diseases" will be given at Mount Sinai Hospital, New York, October 11-15, under the auspices of the American College of Physicians. Dr. Arthur M. Master and Dr. Charles K. Friedberg will direct the course.

The membership of the National Gastroenterological Association was transferred to the American College of Gastroenterology at a meeting held in New York recently. The College will hold its first convention in Washington, D. C., October 25-27 in conjunction with the 19th annual convention of the association. Information on membership may be obtained from the executive secretary of the College, 33 West 60th Street, New York 23.

The National Gastroenterological Association announces that its sixth annual course in postgraduate gastroenterology will be given at the Shoreham, Washington, D. C., October 28-30, under the co-chairmanship of Dr. Owen H. Wangensteen and Dr. I. Snapper. Information may be secured from the association, Department GSJ, 33 West 60th St., New York 23.

The 1954 convention of the National Society for Crippled Children and Adults will be held at the Statler Hotel, Boston, November 3-5.

The second annual meeting of the Inter-Society Cytology Council will be held in Boston, November 12 and 13. Those having material to present are invited to submit three copies of the title and an abstract of 200 words to Dr. John B. Graham, 32 Fruit Street, Boston, before July 15. Complete information may be secured from the secretary-treasurer, Inter-Society Cytology Council, 634 North Grand, St. Louis.

A.M.A. PROGRAM ON TV

Highlights of the A.M.A. annual session in San Francisco will be presented over television on Thursday, June 24, as part of the "March of Medicine" series of telecasts. Sponsored by Smith, Kline and French Laboratories in co-operation with the A.M.A., the telecast will be carried over the N.B.C. network at 9:00 p.m., replacing the Martin Kane show regularly seen at that time.

Committees for 1954-1955

Allied Groups

L. F. Schmaus, Iola, Chr.; C. M. Alderson, Dodge City; W. R. Beine, Coffeyville; C. H. Benage, Pittsburg; R. D. Dickson, Topeka; G. E. Kassebaum, El Dorado; Harry Lutz, Augusta; R. R. Snook, McLouth.

Anesthesiology

P. H. Lorhan, Kansas City, Chr.; L. L. Bresette, Kansas City; H. J. Brown, Winfield; R. S. McKee, Leavenworth; C. D. McKeown, Wichita; R. T. Parmley, Wichita; E. M. Sutton, Salina; F. C. Taggart, Topeka.

Auxiliary

I. J. Waxse, Oswego, Chr.; E. M. Harms, Wichita; E. R. Millis, Kansas City; C. E. Partridge, Emporia; C. O. West, Kansas City.

Blue Shield Fee Schedule

N. L. Francis, Wichita, Chr. ENT; H. O. Anderson, Wichita, Orthopedics; C. M. Barnes, Seneca, General Practice; W. L. Beller, Topeka, Radiology; D. E. Gray, Topeka, Obstetrics and Gynecology; G. F. Gsell, Wichita, Eye; A. G. Isaac, Newton, Urology; B. I. Krehbiel, Topeka, Pediatrics; W. O. Martin, Topeka, Anesthesiology; E. J. Ryan, Emporia, Internal Medicine; L. L. Saylor, Topeka, Surgery; B. E. Stofer, Wichita, Pathology; H. S. Blake, Topeka, Blue Shield.

Blue Shield Relations

C. S. Joss, Topeka, Chr.; Glen Ashley, Chanute; E. S. Brinton, Wichita; W. E. Brownlee, Hutchinson; O. R. Cram, Larned; P. M. Hulett, Anthony; G. E. Manahan, Lawrence; D. E. McCoy, Oberlin; J. C. Mitchell, Salina; A. L. Nichols, Hiawatha; E. B. Scagnelli, Dodge City; J. E. Seitz, Wakeeney; L. N. Speer, Kansas City; R. M. Thomas, Marysville; S. L. VanderVelde, Emporia; M. W. Wells, Winfield; H. M. Wiley, Garden City.

Child Welfare

W. H. Crouch, Topeka, Chr.; M. S. Boyden, Lawrence; D. R. Davis, Emporia; H. P. Jubelt, Manhattan; G. M. Martin, Topeka; F. L. Menehan, Wichita; H. C. Miller, Kansas City; E. G. Padfield, Salina; E. T. Siler, Hays; L. N. Speer, Kansas City.

Conservation of Eyesight

H. E. Morgan, Newton, Chr.; B. J. Ashley, Topeka; F. N. Bosilevac, Kansas City; L. L. Calkins, Kansas City; D. O. Howard, Wichita; M. S. Lake, Salina; D. T. Loy, Great Bend; W. M. Scales, Hutchinson; D. P. Trimble, Emporia; D. D. Vermillion, Goodland.

Conservation of Hearing and Speech

G. O. Proud, Kansas City, Chr.; C. W. Armstrong, Salina; E. L. Gann, Emporia; C. L. Gray, Wichita; J. H. Johnson, El Dorado; C. R. Kempthorne, Manhattan; W. D. Pitman, Pratt; M. J. Rucker, Sabetha; M. J. Ryan, Kansas City; L. B. Spake, Kansas City.

Constitution and Rules

A. W. Fegtly, Wichita, Chr.; W. M. Brewer, Hays; L. D. Johnson, Mission; L. C. Joslin, Harper; C. T. Ralls, Winfield; C. E. Vestle, Humboldt; Clyde Wilson, Emporia.

Control of Tuberculosis

A. L. Ashmore, Wichita, Chr.; Andre Baude, Topeka; H. L. Bogan, Baxter Springs; R. I. Canuteson, Lawrence; Vale Page, Plainville; Charles Pokorny, Halstead; W. G. Rinehart, Pittsburg; C. F. Taylor, Norton; F. A. Trump, Ottawa; C. J. W. Wilen, Manhattan.

Emergency Medical Care

D. P. Trees, Wichita, Chr.; W. H. Algie, Kansas City; G. W. Hammel, El Dorado; P. B. Leffler, Pittsburg; J. W. Manley, Kansas City; J. M. Mott, Topeka; W. A. Smiley, Jr., Junction City; J. F. Thurlow, Hays; Nathaniel Uhr, Topeka.

Endowment

J. W. Randell, Marysville, Chr.; S. G. Ashley, Chanute; J. O. Austin, Garden City; L. J. Beyer, Lyons; C. V. Black, Pratt; H. O. Bullock, Independence; T. P. Butcher, Emporia; V. E. Chesky, Halstead; L. H. Coale, Kansas City; W. M. Cole, Wellington; W. S. Fast, Atchison; R. W. Fernie, Hutchinson; J. L. Grove, Newton; D. G. Holcomb, Liberal; Dwight Lawson, Topeka; G. D. Marshall, Colby; B. P. Meeker, Wichita; J. C. Mitchell, Salina; M. C. Ruble, Parsons; H. B. Russell, Great Bend; E. A. Smiley, Junction City; G. I. Thacher, Waterville; N. V. Treger, Topeka; S. L. VanderVelde, Emporia.

Expert Testimony

C. E. Joss, Topeka, Chr.; E. J. Frost, Wichita; J. L. Lattimore, Topeka; C. D. McKeown, Wichita.

General Practice Award

H. M. Glover, Newton, Chr.; C. W. Bowen, Topeka, Pres. Kansas Acad., Vice-Chr.; G. E. Burket, Jr., Kingman; J. P. Haigler, Hays; Lawrence E. Leigh, Overland Park; F. N. White, Russell.

History

R. R. Melton, Marion, Chr.; W. M. Mills, Topeka, Vice-Chairman; W. L. Anderson, Atchison; A. E. Bair, Independence; H. C. Clark, Wichita; C. C. Nesselrode, Kansas City; A. K. Owen, Topeka; L. L. Saylor, Topeka; M. O. Steffen, Great Bend; C. F. Taylor, Norton.

Hospital Survey

J. H. A. Peck, St. Francis, Chr.; A. C. Armitage, Hutchinson; P. L. Beiderwell, Belleville; W. J. Biermann, Wichita; A. P. Cloyes, El Dorado; M. J. Cox, Dodge City; A. P. Gearhart, Wichita; E. R. Gelvin, Concordia; L. C. Hays, Cedar Vale; J. L. Jenson, Colby; L. W. Patzkowsky, Kiowa; P. A. Pettit, Paola; A. J. Rettenmaier, Kansas City; F. C. Shepard, Clay Center; C. D. Snyder, Winfield.

Industrial Medicine

M. A. Walker, Kansas City, Chr.; J. W. Cavanaugh, Topeka; J. A. Grove, Newton; C. W. Hall, Hutchinson; H. R. Hodson, Wichita; P. C. Nohe, Kansas City; H. L. Regier, Kansas City; R. W. Urie, Parsons.

Maternal Welfare

D. E. Gray, Topeka, Chr.; D. A. Anderson, Salina; R. M. Carr, Junction City; L. E. Filkin, Concordia; H. M. Floersch, Kansas City; H. M. Foster, Hays; R. G. Heasty, Manhattan; R. L. Hermes, Lawrence; G. M. Martin, Topeka; R. L. Newman, Kansas City; C. D. Shrader, Newton; F. L. Smith, Jr., Colby; R. A. West, Wichita.

Medical Assistants

L. G. Allen, Kansas City, Chr.; R. A. Crawford, Hutchinson; H. J. Davis, Topeka; A. E. Hiebert, Wichita; G. R. Peters, Kansas City; J. M. Porter, Concordia; Walter Stephenson, Norton.

Medical Economics

L. S. Nelson, Jr., Salina, Chr.; J. N. Blank, Hutchinson; R. L. Gench, Fort Scott; E. J. Grosdidier, Kansas City; D. H. Macrae, Topeka; G. E. Milbank, Wichita; M. B. Miller, Topeka; F. A. Moorhead, Neodesha; B. A. Nelson, Manhattan; R. Solhberg, Jr., McPherson.

Medical Schools

J. B. Fisher, Wichita, Chr.; R. G. Ball, Manhattan; C. M. Barnes, Seneca; R. M. Carr, Junction City; R. D. Dickson, Topeka; A. C. Hatcher, Wellington; G. R. Peters, Kansas City; R. C. Polson, Great Bend; R. E. White, Garnett; G. G. Whitley, Douglass; H. H. Jones, Jr., Winfield.

Mental Health

A. J. Adams, Wichita, Chr.; H. V. Baer, Parsons; R. L. Drake, Wichita; D. B. Foster, Topeka; T. L. Foster, Halstead; Mary Glassen, Phillipsburg; E. D. Greenwood, Topeka; L. W. Hatton, Salina; C. C. Hawke, Winfield; George Jackson, Topeka; C. J. Kurth, Wichita; W. F. Roth, Jr., Kansas City; D. R. Wall, Wichita; M. E. Wright, Lawrence.

Necrology

J. F. Gsell, Wichita, Chr.; L. J. Brethour, Junction City; A. C. Gulick, Goodland; F. D. Lose, Madison; Alfred O'Donnell, Ellsworth; A. E. Titus, Cottonwood Falls.

Nominations

C. H. Benage, Pittsburg, Chr.; W. F. Bernstorf, Winfield; O. W. Davidson, Kansas City; J. H. A. Peck, St. Francis; H. N. Tihen, Wichita.

Past Presidents' Advisory Committee

All past presidents of The Kansas Medical Society.

Pathology

J. L. Lattimore, Topeka, Chr.; T. R. Hamilton, Kansas City; C. A. Hellwig, Halstead; N. P. Sherwood, Lawrence; B. E. Stofer, Wichita.

Postgraduate Study

E. L. Mills, Wichita, Chr.; W. H. Algie, Kansas City; M. H. Delp, Kansas City; H. L. Heibert, Topeka; H. H. Jones, Winfield; H. P. Palmer, Scott City; Maurice Snyder, Salina.

Public Policy

J. L. Lattimore, Topeka, Chr.; all past presidents of The Kansas Medical Society.

Public Relations

E. S. Brinton, Chr., Wichita; J. F. Barr, Ottawa; H. S. Blake, Topeka; T. P. Butcher, Emporia; G. W. Cramer, Parsons; L. G. Heins, Abilene; J. D. Hilliard, Medicine Lodge; N. E. Hull, Hays; M. S. Lake, Salina; P. B. Leffler, Pittsburg; J. W. Manley, Kansas City; C. W. Miller, Wichita; V. R. Moorman, Hutchinson; R. H. O'Neil, Topeka; C. O. Stensaas, Arkansas City; V. E. Wilson, Kansas City.

Rural Health

V. E. Brown, Sabetha, Chr.; J. R. Bradley, Greensburg; J. G. Claypool, Howard; C. S. Fleckenstein, Onaga; E. T. Gertson, Atwood; R. E. Grene, La Crosse; C. C. Gunter, Quinter; K. R. Hunter, Lebo; D. L. Marchbanks, Hill City; John Neuenschwander, Hoxie; R. H. O'Donnell, Ellsworth; J. W. Parker, Burlington; E. F. Steichen, Lenora; R. P. Stoffer, Halstead; G. A. Surface, Ellis; H. J. Williams, Osage City.

Stormont Medical Library

J. D. Beck, Topeka, Chr.; G. F. Corrigan, Wichita; Rodger Moon, Prairie Village; A. K. Owen, Topeka; W. L. Valk, Kansas City.

Study of Heart Disease

P. W. Morgan, Emporia, Chr.; D. R. Bedford, Topeka; P. M. Clark, Independence; E. G. Dimond, Kansas City; C. W. Erickson, Pittsburg; H. A. Flanders, Hays; L. H. Leger, Kansas City; G. L. Norris, Winfield; L. O. Peckenschneider, Halstead; D. C. Wakeman, Topeka.

Venereal Disease

M. D. McComas, Jr., Concordia, Chr.; M. L. Bauman, Wichita; A. B. Harrison, Wichita; H. F. O'Donnell, Wichita; G. S. Voorhees, Leavenworth; V. M. Winkle, Topeka.

ACTIVITIES OF MEMBERS

Dr. Bruce A. Higgins, Sylvan Grove, who has been practicing in Lincoln County since the 1920's, announced his retirement last month because of failing health and moved to Topeka to make his home with his son.

Four physicians from the Menninger Foundation, Topeka, took part in the program of the American Psychiatric Association at a meeting held in St. Louis last month. Dr. Edward D. Greenwood was chairman of a sectional meeting on child psychiatry and participated in a discussion of "The Care of the Emotionally Disturbed Child." Dr. Will Menninger was chairman of meetings of the committees on "Community Aspects of Psychiatry." Dr. Karl Menninger led a discussion on "The Psychotherapy of the Impulsive Personality," and Dr. Lewis L. Robbins took part in a discussion of "Current Trends in the Collaboration of Psychiatry and Social Work."

Dr. Rodney G. Carter, Dr. Edgar L. Robinson, Dr. Charles E. Gollier, and Dr. William G. Chappuie presented a forum on diseases of children before a public meeting in Independence last month under the sponsorship of the Independence Rotary Club.

Dr. Adolph Mueller, president of the Leavenworth County Medical Society, described progress in medicine at a town meeting in Leavenworth last month.

Dr. W. E. Michener, Topeka, was honored recently by Capitol Post No. 1 of the American Legion for his "long and unselfish service to disabled veterans." He was presented with an inscribed plaque recognizing 30 years of service.

Dr. L. D. Johnson, who has practiced medicine for 52 years, 48 years in Chanute, closed his office recently and moved to Mission.

Dr. Arnold H. Baum and Dr. R. J. Ohman presented part of a program for a meeting of nurses held at Dodge City in April. They participated in a discussion on "The Care of the Infant."

Dr. Frank A. Moorhead, Neodesha, spoke on tuberculosis over radio station KCRB at Chanute recently.

Dr. Ira I. Smith, Altanta physician for 41 years, was guest of honor at a community-wide reception held in April. He has also served the city as mayor for several terms.

Dr. Donald D. Dieter, Salina, addressed a group of cancer workers in Dickinson County in Abilene during the cancer campaign.

Dr. Francis J. Bice, who has been in the Navy during the past two years, has returned to his home and practice in Wakeeney.

Dr. Porter Barbera, Independence, spoke on "Anemia" before a meeting of the Independence R.N. Club recently.

Dr. John W. Jacks, Pratt, who has been stationed

at Corpus Christi, Texas, since January, 1953, when he entered military service, has returned to Pratt to practice in association with Dr. M. E. Christmann.

Dr. William J. Reals and Dr. Alfred M. Tocker, Wichita, took part in the program for the annual convention of the Kansas Society of Medical Technologists, held at Wichita last month.

Dr. Wendell L. Good, formerly of Perry, has announced the opening of an office in Mission.

Dr. Martin J. FitzPatrick, of the University of Kansas Medical Center, was speaker at the annual dinner meeting of the Wyandotte County Tuberculosis and Health Association in Kansas City. His subject was "Unsolved Problems of Tuberculosis."

Dr. Kenneth L. Knuth, who has been serving in the Navy since October of 1952, has returned to his practice in Atwood.

Dr. Roy C. Knappenberger, Wichita, recently became a diplomate of the American Board of Pediatrics.

Dr. James Robert Weaver has received his discharge from the Army and has returned to practice in Wichita.

A feature story about Dr. Claude E. Burtch, Portis, was published in the *Smith Center Pioneer* on May 6 in tribute to his 50 years of practice.

Dr. Edward D. Greenwood, Topeka, was named last month to head the National Advisory Council on State and Local Action for Children and Youth. The council is the interim group which will follow up the 1950 Mid-Century White House Conference on Children and Youth until the White House calls the next conference in 1960.

Dr. James B. Weaver, Kansas City, was named president of the Mid-Central Orthopedic Society at a meeting held in Kansas City recently. Dr. H. O. Anderson, Wichita, was elected secretary-treasurer, and Dr. C. L. Francisco, Kansas City, was chosen chairman of a committee on arrangements.

Dr. Dale U. Loyd, Wichita, has completed the examinations of the American Board of Anesthesiology and is now a diplomate of the board.

Dr. D. C. Niederluecke, Valley Falls, discontinued practice there early in May and is practicing temporarily in Appleton City, Missouri. In July he will begin a three-year residency in obstetrics and gynecology.

Dr. Jerome S. Menaker, Wichita, was one of the speakers at a sectional meeting of the Academy of Obstetrics and Gynecology in Oklahoma City in April.

Dr. Glenn R. Peters, Kansas City, has been named to represent the Wyandotte County Medical Society on a city council formed to conduct a clean-up project.

Dr. J. Gordon Claypool, Howard, and Dr. George E. Burket, Jr., Kingman, have been named to present an exhibit sponsored by the Kansas Chapter, American Academy of General Practice, at the American Medical Association meeting to be held in San Francisco late this month.

Dr. Carroll D. Behrhorst, who has been serving in the Navy during the past 18 months, received his discharge recently and has returned to practice in Winfield.

Dr. Frederick P. Wolff, Pratt, was named last month as health officer for Pratt County in the reestablishment of a health department there after a lapse of six months.

Award to Mrs. Leo J. Schaefer

Mrs. Leo J. Schaefer, Salina, who will complete her term of office as president of the Woman's Auxiliary to the American Medical Association this month, was one of 15 individuals honored by the American Medical Education Foundation recently for "outstanding contributions to the preservation and continuance of the high standards of medical education in the United States of America." She was given an "Award of Merit."

Seventeen organizations also received the award, and included in the number were the A.M.A. and its woman's auxiliary.

Amendments to Constitution and By-Laws

Since numerous amendments to the Constitution and By-Laws of The Kansas Medical Society were approved by the House of Delegates in both 1953 and 1954, the constitutional booklets distributed to the membership in 1952 are no longer completely accurate. An effort is now being made to present all changes in a form that will enable members of the Society to keep their own constitutional booklets up to date.

The paragraphs below, printed in the order in which they appear in the booklet, are to be substituted for the articles, chapters, and sections of the same numbers.

CONSTITUTION

ARTICLE VI.—Council.

Section 1. The Council shall consist of one Councilor from each councilor district, and in addition the officers of the Society, chairman of the Editorial Board and the delegates to the American Medical Association as ex-officio members.

ARTICLE VII—House of Delegates.

Section 1. The House of Delegates shall be the primary legislative and governing body of this Society, and shall consist of the duly elected delegates and the officers, councilors, chairman of the Editorial Board and past presidents of this Society.

BY-LAWS

CHAPTER I.—Membership

Section 4. Members of this Society may be enrolled as honorary members upon the certified recommendation of the component societies to which they belong. Such recommendation shall be based upon years of faithful service in the medical profession, or upon other grounds acceptable to the Council. Honorary members shall be entitled to all of the benefits and privileges of active members, but shall be exempt from the payment of assessments.

CHAPTER II.—Assessments.

Section 1. The amount of the annual assessment for The Kansas Medical Society only shall be not more than fifty dollars per member, the exact amount to be determined by the Council after consideration of the annual budget for the ensuing year and to be announced to the various component societies not less than three months before the beginning of each fiscal year. Such assessments shall be levied against and paid by the component societies in the manner provided by this Constitution and By-Laws, except that any new members of this Society being accepted by a component society after July 1, shall be assessed one-half the sum decided upon by the Council, and shall be accorded all the rights and benefits of this Society, including defense, until the succeeding January 1.

CHAPTER VI.—Election of Officers.

Section 1. A Nominating Committee of five shall be selected by ballot from all living past presidents, still members of this Society, at the last meeting of the House of Delegates of each annual session; the committee to consist of the five receiving the greatest number of votes and in case of tie vote on the fifth member the committee will consist of six instead of five. The past president receiving the greatest number of votes shall be the chairman. No past president may serve on more than two consecutive annual committees; the committee shall meet not later than 90 days prior to the next annual session and present for publication in the Journal a list of candidates for each elective office consisting of one or more candidates for the offices of president-elect, first vice-president, secretary, treasurer, and delegate-elect to the American Medical Association, and three or more candidates for the offices of second vice-president and alternate delegate to the American Medical Association. Whenever possible or practical, the candidates for treasurer should be named from residents of the city or vicinity of the location of the central office.

CHAPTER VIII.—The Council.

Section 12. A majority of the members of the Council shall constitute a quorum.

CHAPTER VIII.—The Council.

Section 13. The councilor districts shall be comprised of the following counties:

District 1—Atchison, Brown, Doniphan, Jackson, Jefferson, Leavenworth, Marshall and Nemaha.

District 2—Wyandotte County.

District 3—Anderson, Douglas, Franklin, Johnson, Linn and Miami.

District 4—Allen, Bourbon, Cherokee, Crawford, Labette, Montgomery, Neosho, Wilson and Woodson.

District 5—Clay, Geary, Pottawatomie, Riley and Washington.

District 6—Shawnee County.

District 7—Chase, Coffey, Lyon, Morris, Osage and Wabaunsee.

District 8—Butler, Chautauqua, Cowley, Elk and Greenwood.

District 9—Cloud, Dickinson, Ellsworth, Jewell, Lincoln, Mitchell, Ottawa, Republic and Saline.

District 10—Harvey, McPherson, Marion, Reno and Rice.

District 11—Sedgwick County.

District 12—Barber, Harper, Kingman, Pratt and Sumner.

District 13—Ellis, Graham, Osborne, Phillips, Rooks, Russell, Smith and Trego.

District 14—Barton, Edwards, Hodgeman, Ness, Pawnee, Rush and Stafford.

District 15—Clark, Comanche, Ford, Gray, Kiowa, Meade and Seward.

District 16—Cheyenne, Decatur, Gove, Logan, Norton, Rawlins, Sheridan, Sherman, Thomas and Wallace.

District 17—Finney, Grant, Greeley, Hamilton, Haskell, Kearny, Lane, Morton, Scott, Stanton, Stevens and Wichita.

CHAPTER IX.—Defense Board.

Section 1. Members of The Kansas Medical Society are urged to carry individual professional insurance with commercial companies as these usually include possible indemnity in their policies, but additional assistance of the Society defense counsel may be provided if necessary or desired. It shall be the duties of the members of the Defense Board, severally or collectively, to investigate all claims of malpractice made against members desiring Society assistance; to assist in conducting the defense; to assist such member in planning for defense and if requested by the defendant and deemed advisable by the Defense Board procure the services of an attorney approved by The Kansas Medical Society, provided the entire cost of such services shall be paid by the defendant. The Society shall not be obligated to pay any fees for legal services, nor any costs or judgments arising out of any action involving the professional services of any member.

CHAPTER IX.—Defense Board.

Section 6. Disbursements, for defense to include reimbursement for any actual expenses incident to travel, by members of the Defense Board, required in the performance of official duties, and a per diem sum fixed by the Council for the days actually spent in such official duties may be allowed. Bills for defense expenditure, authorized by the Defense Board, and approved by the chairman, shall be paid by vouchers signed by the treasurer and countersigned by the president and secretary, and charged against the Defense Fund for that year. In the event an insufficient sum is budgeted, the treasurer shall be empowered to provide, upon a sufficient accounting, an extra sum not to exceed $300, and if additional amounts are necessary authorization shall be secured therefore from the Council. Any surplus at the end of the fiscal year shall be considered in the defense budget for the succeeding year.

CHAPTER XI.—Committees. Section 1a

Committee on Public Relations Sec. 29
Past Presidents Advisory Committee Sec. 35
Committee on Pathology Sec. 36
Committee on General Practice Award Sec. 37

CHAPTER XI—Committees.

Section 7. The Committee on Arrangements shall be appointed by the component society of the county in which the annual session is to be held, and shall consist of as many sub-committees as may be desired. It shall be the duty of this committee to provide suitable accommodations for the meeting places of this Society, the Council, the House of Delegates and their respective committees. It shall prepare and arrange for diversified scientific programs subject to the approval of the Executive Committee, who shall determine that the anticipated expenses of the meeting are within the budget and shall have general charge of all the annual session arrangements, in co-operation with the executive secretary. Its chairman shall report an outline of the arrangements and program to the executive secretary for publication in the annual session program, and shall make additional announcements concerning the session as occasion may require.

CHAPTER XI.—Committees.

Section 29. The Committee on Public Relations shall consist of five or more members who shall, through the media of speakers' bureaus, newspapers, radio, and personal and group meetings, make material and information available to the public on all subjects for betterment of general health, and secure for the profession criticisms and valuable information for the improvement of relations of physicians individually and collectively with patients and the public generally. At least the chairman and one other member of this committee shall have served on the retiring committee.

CHAPTER XI.—Committees

Section 35. The Past Presidents Advisory Committee shall be composed of *all living* past presidents. Meetings shall be held at a convenient time and place at the call of the immediate past president, and succeeding meetings as desired. The chairman and secretary are to be selected at the first meeting of each year.

The purpose of the committee shall be mutual fellowship and means of free discussion of all pertinent matters affecting the ideals, aims, proficiency, and successful operation and work of The Kansas Medical Society. The advice and recommendations arising from these meetings and discussion, which have the benefit of experience in office, shall be communicated to the Council through the immediate past president who is their official representative upon that Council.

CHAPTER XI.—Committees

Section 36. The Committee on Pathology shall be composed of five pathologists, appointed for a term of three years. Originally, one shall be appointed for one year, two for two years, and two for three years. All matters presented to the Society relating to the practice of pathology shall be referred to this committee for study and recommendation.

CHAPTER XI.—Committees.

Section 37. The Committee on General Practice Award shall consist of five or more members of which one shall be a councilor and one the president of the Kansas Academy of General Practice or a member of that academy designated by him. Two or more members shall have served on the retiring committee. The duties of the committee shall be to determine an individual member and submit adequate material and information in support of any Kansas Medical Society member desired to be submitted to the American Medical Association committee as a candidate for the annual general practitioner's award.

CHAPTER XII.—Component Societies.

Section 5. Each component society shall judge the qualifications of its own members, but as these societies are the only portals of entrance to this Society and to the American Medical Association every reputable and ethical physician having a degree of Doctor of Medicine from an accredited medical school and licensed by the Kansas State Board of Medical Registration and Examination, legally registered in his county of practice, and the greater proportion of his professional work be conducted within this state, shall be privileged to apply for membership. Before a charter is issued to any component society full and ample notice and opportunity to become a member shall be given to every physician in that county who is eligible as herein provided.

CHAPTER XII.—Component Societies.

Section 7. Any physician who may feel aggrieved by the action of the component society of his county in refusing him membership, or in suspending or expelling him, shall have the right to appeal to the Council.

CHAPTER XII.—Component Societies.

Section 8. In hearings of appeals, the Council may admit oral or written evidence as in its judgment will best and most fairly present the facts, but in every case of appeal efforts for conciliation and compromise shall be the responsibility or duty of the councilor of that district, and shall precede all hearings.

CHAPTER XII.—Component Societies.

Section 9. A physician living on or near a county line within this state, and practicing in adjacent counties, may hold membership in the county of the major proportion of his professional work, or in the county most convenient for him to attend if permission is obtained from the component society in whose jurisdiction he resides and by election to the component society in which he desires membership.

CHAPTER XII.—Component Societies.

Section 10. On and after the passage of this amendment, no physician shall be admitted to new membership in this Society, or in any of its component societies, who is a member of a state or component medical society of another state unless he intends by his application to transfer membership and the major portion of his professional work to this state. Any physician residing in a county adjacent to another state or in a county of an adjoining state, licensed to practice in both states, and carrying on his professional work in both states, but the greater part of his professional work is carried on in this state, shall be eligible for membership in The Kansas Medical Society. A full time teaching assignment at the University of Kansas School of Medicine or in any state supported school, college or institution shall be considered the major portion of his professional work, regardless of the proportion of financial remuneration received from professional work outside the state of Kansas. When there are unusual circumstances connected with the above limits of eligibility which cause his membership to be advisable in this state or the adjoining state, consent to membership must be obtained from the county or state society of his residence.

BLUE SHIELD

Participation by Kansas Doctors

Representation of Blue Shield participating physicians was excellent at both the Blue Cross and Blue Shield annual board meetings held in May.

Dr. L. W. Reynolds of Hays was elected president of Kansas Blue Shield at the annual meeting of that group held in Topeka on May 2. He succeeds Dr. H. S. Blake of Topeka.

Dr. Reynolds has been a member of the Blue Shield board for three years and last year served the organization as vice-president. He has been a Blue Shield participating physician since the plan was established in this state in 1946.

Other Blue Shield officers elected at the annual meeting were: Dr. Francis T. Collins, Topeka, vice-president; Dr. R. P. Watterson, McPherson, sec-

retary-treasurer; and Dr. L. E. Filkin, Concordia, executive vice-president.

Dr. E. J. Ryan, Emporia; Dr. James B. Fisher, Wichita, and Dr. Floyd L. Smith, Colby, are new members of the Blue Shield board this year. Dr. Filkin is a re-elected member of the board.

Special recognition was paid to Dr. Dwight Lawson, Topeka, a retiring member of the board. Dr. Lawson has served on the Blue Shield board for seven years and is a past president of the plan.

BLUE CROSS BOARD

Twelve Blue Shield participating physicians are members of the Blue Cross board of directors at all times. This board is composed of equal representation of the member public, hospitals, and physicians, and beginning this year these representatives were elected from the local districts.

The physician representation elected this year includes Dr. Cecil C. Hunnicutt, Sabetha; Dr. Ralph E. White, Garnett; Dr. Glen Ashley, Chanute; Dr. Thomas L. Foster, Halstead; Dr. H. St. Clair O'Donnell, Ellsworth, and Dr. J. L. Lattimore, Topeka.

Other physicians on the board whose terms extend for at least another year are Dr. A. L. Ashmore, Wichita; Dr. Thomas P. Butcher, Emporia; Dr. Thomas L. Hill, Arkansas City; Dr. G. E. Kassebaum, El Dorado; Dr. H. Preston Palmer, Scott City, and Dr. J. H. A. Peck, St. Francis.

Dr. Lattimore was re-elected president of Kansas Blue Cross at that organization's annual meeting May 13. He begins his sixth full term as president of the Blue Cross board. Other re-elected officers of the Blue Cross board are C. O. Wright, first vice-president, Topeka; Herman J. Andres, second vice-president, Newton; W. Laird Dean, treasurer, Topeka, and Warren M. Crosby, secretary, Topeka.

THE KANSAS PRESS LOOKS AT MEDICINE

Editor's Note. In this section the JOURNAL *reproduces editorials relating to medicine which have appeared in the lay press. An effort is made to include both favorable and unfavorable comments, and the Editorial Board in no instance assumes responsibility for the opinions expressed.*

SOMEBODY ELSE'S DOCTOR

"Doctors are more concerned with their pocketbooks than their patients," said a physician, speaking before a meeting of members of his own profession this week. " 'Knife-happy' surgeons perform many unnecessary operations on women," declared the head of a hospital staff at a session of his fellows.

What merit there may be to these harsh criticisms from within, I don't know. But there have been enough of them in these and related veins by distinguished doctors lately to indicate that the medical profession is beginning to do some soul-searching and self-policing.

It was about time. In a scientific way the healing profession has made tremendous strides. But it has been woefully weak on its personal and public relations. Fortunately it is now beginning to appreciate that itself.

Your doctor or my doctor is above reproach, or he wouldn't enjoy that relationship. But somebody else's doctor has been acting in a way to spread impressions that are as unfortunate for the profession as they are for the public.

Somebody else's doctor has become so obsessed with the possibility of socialized medicine that he has become more propagandist than physician. Somebody else's doctor has accumulated so many patients that he can treat them only like laboratory specimens. Somebody else's doctor has become so immersed in new methods and machinery of treatment that he has become a machine himself. Somebody else's doctor has acquired such a feeling of omniscience that he too frequently takes over when nature would take a better course.

As a layman diagnoses these ailments, however, no operation is called for, nor even miracle drugs. Only a little applied psychology.

The bedside manner has disappeared with the Old Family Doctor who had it. What the medical scientist who has succeeded him must acquire, not only for the sake of public relations but also to make his talents fully effective, is clinic chumminess and hospital hospitality.—*Hutchinson News-Herald, March 27, 1954.*

SOUND SOLUTION

The President's message to Congress on health matters has again focused national attention on the problem of extending voluntary health insurance coverage and still keeping the cost within the means of the average family.

Without debating the President's proposals, there is one sound way of doing that—and it is a way which would involve no revolutionary experiment. It lies in legislation which would permit income tax deductions of medical and hospital bills, and premiums paid for health insurance. Then individuals would no longer pay heavy taxes on money which simply passed through their hands, and had to be spent for unavoidable expenses completely beyond their contro.—*El Dorado Times, February 18, 1954.*

The Anticoagulants: Heparin and Dicumarol

Clarence E. Thompson, M.D.

Kansas City, Missouri

INTRODUCTION

Since the discovery of the anticoagulants, dicumarol and heparin, there has been a progressive increase in their application in various disease states. It is not within the scope of this paper to discuss the various disease states and indications for anticoagulant therapy. Rather, here I shall discuss the basic principles of the anticoagulant properties to clarify the physiological tenets of their usage. While there have been many anticoagulants, heparin and dicumarol in clinical usage have been found the best of those known to date, and, although they are not entirely satisfactory, their usage has become quite widespread.

The specifications of an entirely satisfactory anticoagulant are: (1) an agent having a predictable effect on the coagulation of blood; (2) an agent that does not have any harmful side effects; (3) its effects should be rapidly neutralized by a potent antagonistic medication readily available; (4) the agent should be effective via the oral route; (5) it should be possible to measure the anticoagulant effect easily at bedside, and (6) the agent should not be expensive.[1]

HEPARIN: DISCOVERY, SITE, AND MODE OF ACTION

Heparin was first recorded as having an anticoagulant action by J. MacLean, a medical student at Johns Hopkins University, in 1916.[6] The original substance was heparphosphatide prepared from dog liver.[2] Little was done for the next few years until 1929, when Best of Toronto became interested. After four years of experimentation, a purified form of heparin was isolated from ox lung in 1933.[3]

The exact site of the action of heparin in the clotting mechanism is not known,[4] but it is known that both phases of the mechanism are inhibited by heparin.[7] To better explain it is necessary to graphically show the clotting mechanism.

Prothrombin + CA + Thromboplastin → Thrombin

Thrombin + Fibrinogen → Fibrin

Heparin acts to inhibit the formation of thrombin and also the formation of fibrin.[7]

Heparin inhibits coagulation of blood in vivo and in vitro by at least four distinct actions: (1) It retards rate of prothrombin conversion to an extent that is inversely proportional to the amount of thrombokinase present; (2) It alters the effectiveness of the thrombin formed by combining with the albumin co-factor to form a complex that binds thrombin; (3) It inhibits platelet agglutination, and (4) It inhibits fibrin deposition.[5]

The latent period of heparin action on blood coagulation is short, whereas its latent period of action on platelet agglutination is long, sometimes several hours. This is important because at focus of injury, platelets accumulate and agglutinate, and subsequently fibrin is deposited about and within the clumps of platelets with resultant thrombus formation. It is postulated that the platelet agglutination inhibition is due to effect of heparin by its strong electrical charge to alter the electric potentials or surface tension of the platelets.[3]

Later studies have disclosed more of the pharmacology of heparin. It is now known that heparin is a complex organic polymer, that it has the structure of a mucoitin poly sulfuric acid, and that it contains a basic tetrasaccharide unit. This tetrasaccharide is esterified with possibly a variable number of sulfuric acid groups resulting in a strongly acidic and electronegative compound which has the property of forming stable salts with proteins. This has been postulated as an explanation of its anticoagulant properties inasmuch as it may modify the action of the protein enzymes concerned in the coagulation of blood. Heparin in itself is only weakly if at all anticoagulant, as may be demonstrated on purified clotting factors of blood. In the presence, however, of a co-factor of normal plasma or serum, heparin is a powerful anticoagulant. This co-factor, although not definitely identified, is in the most soluble of the serum albumin fractions.[7]

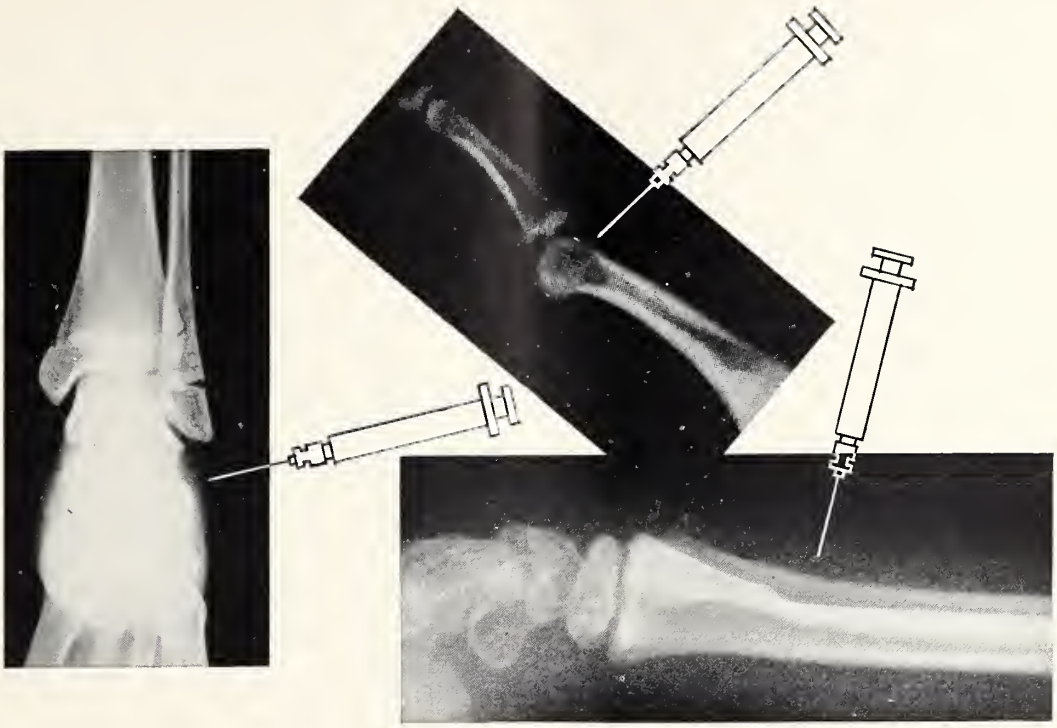
It is interesting to note that in a study of 67 normal fasting subjects, some of whom had had surgical procedures, that there was little alteration in co-factor activity of serum when no thromboembolic complications occurred; when thromboembolic complications did occur, there was an appreciable reduction in co-factor activity.[8]

PREPARATIONS, DOSAGES AND ROUTES OF ADMINISTRATION

With the advance of knowledge of heparin, various preparations and methods of administration have developed. Heparin cannot be given orally.[1] There are several methods of heparin administration.

This is one of 11 theses, written by fourth year students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group judged to be the best by the faculty at the school.

Thesis written while the author was a fourth year student. Dr. Thompson is now interning at Menorah Hospital, Kansas City, Missouri.



Use of Alidase® in Closed Wounds: Contusions, Sprains, Dislocations, Simple Fractures

In traumatic surgery[1] where "definitive treatment . . . is often delayed while the surgeon waits for nature to dispose of hematoma and oedema" Alidase is an efficient means[1, 2] of accelerating dispersion of accumulated fluids.

Swenson[2] has described his highly successful results with Alidase in various types of closed wounds. He summarized them as follows:

To remove local fluid accumulations in contusions or bruises, "The usual dose, 500 viscosity units Alidase® mixed in a small amount of normal saline, is injected into the localized fluid. Mixing the hyaluronidase in 1 per cent procaine solution will also produce local vasodilatation, relief of local pain and more rapid absorption of the fluid mass. This method can also be applied to traumatized bursae or synovial spaces which do not respond to repeated aspirations."

The point of maximal pain is infiltrated with 10 cc. of a 1 per cent procaine solution to which 500 viscosity units of Alidase have been added. With this simple technic, a high percentage of successful results has been obtained.

Alidase may be used to advantage to produce more rapidly a short-acting, complete block anesthesia and to facilitate reduction in subluxation or complete dislocations of the interphalangeal joints. When anesthesia is required for fracture reduction, local block anesthesia can be simplified by adding Alidase to the anesthetic solution. Alidase also tends to decrease local edema and hematoma formation.

Fluids administered with Alidase are rapidly absorbed from subcutaneous tissue. The simplicity of hypodermoclysis avoids the cumbersome arm board, permits convenient administration with little or no pain or swelling, is vein-sparing and saves nursing time in such conditions as burns, postoperative states, toxemias and parenteral alimentation.

Alidase (brand of hyaluronidase) is supplied in serum-type ampuls of 500 viscosity units. It is accepted by the Council on Pharmacy and Chemistry of the American Medical Association. G. D. Searle & Co., Research in the Service of Medicine.

---

1. MacAusland, W. R., Jr.; Gartland, J. J., and Hallock, H.: The Use of Hyaluronidase in Orthopaedic Surgery, J. Bone & Joint Surg. *35-A*:604 (July) 1953.
2. Swenson, S. A., Jr.: Minor Surgical Aspects of Closed Wounds, Am. J. Surg. *87*:384 (March) 1954.

1. Intermittent intravenous, using 10 mg. per cc. Preliminary Lee and White clotting time (LWCT) followed by 50 mg. heparin intravenously if LWCT 10-30 minutes and 4 hours later repeat. If no hyperreaction occurs, continue 50 mg. intravenously every 4 hours, though one night interval may be 6 hours with LWCT every 24 hours. The objective is to maintain LWCT at 25-45 minutes or at least three times the patient's normal LWCT.

2. Continuous intravenous, to be used only if intravenous fluids are required. Use 100 mg. heparin per 500-1000 cc. physiological saline and administer at 40 drops per minute. Perform LWCT after about ½ the intravenous fluid is given, and then judge rate of administration by results obtained at that time.

3. 200-300 mg. heparin in retarding media. Use capsule without adding vasoconstrictors. Clotting time is not always adequately prolonged so LWCT is needed every 8 hours at first. Heparin is effective for 24 hours when given this way.[9]

Intramuscular administration of heparin in Pitkin's menstruum has been used, and ideally this may reduce the frequency of injections to one every 48 hours. It may be painful, inadequate heparinization may result, and a prolonged excessive effect may occur.[10]

Another preparation is Depo-Heparin Sodium (Upjohn) which is 200 mg. of heparin sodium, with or without vasoconstrictors, per cc. of a gelatin-dextrose medium. It appears to be less painful than heparin in Pitkin's menstruum, but acts in a similar manner. It seems to be particularly useful as a means of administering heparin during the first few days of combined therapy. The 200 mg. given intramuscularly twice a day is satisfactory for the majority of patients. The clotting time, determined just before a dose is to be given, should not exceed 20 minutes. If it does, the dose should be postponed for six hours or omitted.[11]

Depo-Heparin has been used clinically with good results. In 15 cases with initial doses of 300-400 mg. and 200 to 300 mg. on successive days, coagulation times of 2-4 times normal resulted. No complications were noted in this series of cases in which Depo-Heparin was used.[13]

Heparin may be given by subcutaneous route but the magnitude of doses required and danger of hematoma render this route impracticable.[5]

Stats and Neuhof state the intravenous administration of heparin, even in interrupted form, is attended by too many difficulties to permit general use. They used a concentrated aqueous solution of heparin, 100 mg. per cc., that was free from foreign substances, given into the gluteal muscles at 8-12 hour intervals with maximum doses of 450 mg. per 24 hours. They obtained good results with coagulation times of 18-24 minutes by the Lee and White technic.[12]

A recent study to ascertain the usefulness of heparin in long term anticoagulant therapy revealed the following results: 37 patients with thrombo-embolic disease were treated with heparin alone up to five weeks. In 9 cases the heparin was given intravenously with the best results being obtained by using 50 to 75 mg. four hourly doses. Intramuscular injections of aqueous solution containing 250 mg. of heparin per cc. were given to 23 patients. In this series, 150 mg. every 12 hours was found sufficient. Hematomas were noted when 150 mg. doses were exceeded. Five patients were given combination intravenous and intramuscular injections. All these methods were found to be successful in maintenance of therapeutic levels over long periods of time.[14]

In the use of heparin it is essential that the therapeutic result be followed by clotting times, and this is a modification of the Lee and White clotting time.

Equipment: Two clean 75 x 8 mm. test tubes. Sterile 5 or 10 ml. syringe, No. 21 needle. The same size tubes should be used for all tests. Procedure: Rinse syringe and tubes with isotonic saline. Draw 6 cc. of blood, noting time at which blood first appears in syringe, remove needle, and gently place 2 cc. of blood in each of tubes. Wait 3 minutes, then gently tilt one tube every 30 seconds until it can be inverted, and do likewise with second tube until it can be inverted. This is the end point.

Precautions: Minimal stasis and clean venipunctures are essential. Test is done at room temperature of 65°-95° F.

Standard: Normal clotting time varies from 8-14 minutes.[15]

CONTRAINDICATIONS, TOXICITY AND TREATMENT OF OVERDOSAGE

The contraindications to the use of heparin are few, and the reasons for them are obvious. They are: (1) blood dyscrasias with bleeding tendency, (2) presence of purpura, (3) subacute bacterial endocarditis due to possibility of purpura, (4) postoperative brain or spinal cord surgery due to drastic effects of minor bleeding. Also heparin should be used with caution in ulcerative lesions or with drainage tubes of any sort.[10]

Hypersensitivity to heparin has been noted in the clinical use of the drug. DeTakats reports that he has seen several patients with increased reactivity to heparin, even though they have never received heparin before. He noted this reactivity may be accompanied by flushing of the face, bronchospasm, edema or urticaria.[16] Gotz reported one patient who developed a severe asthmatic attack after receiving only 10 mg. of heparin intravenously.[17] One patient became cyanotic, developed asthmatic breathing, and had an unobtainable pulse and blood pressure 60 seconds



KARO SYRUP **BELONGS IN THIS PICTURE!**

... a carbohydrate of choice
in milk modification for 3 generations

OPTIMUM caloric balance—60% of caloric intake, gradually achieved in easily assimilable carbohydrates —is assured with Karo. Milk alone provides 28%, or less than half the required carbohydrate intake.

A MISCIBLE liquid, Karo is quickly dissolved, easy to use, readily available and inexpensive.

A BALANCED mixture of dextrins, maltose and dextrose, Karo is well tolerated, easily digested, gradually absorbed at spaced intervals and completely utilized.

PRECLUDES fermentation and irritation. Produces no reactions, hypoallergenic. Bacteria-free Karo is safe for feeding prematures, newborns, and infants—well and sick.

LIGHT and dark Karo are interchangeable in formulas; both yield 60 calories per tablespoon.

CORN PRODUCTS REFINING COMPANY • 17 Battery Place, New York 4, N. Y.

after receiving intravenous injection of 50 mg. of sodium heparin. This patient responded to injection of 0.2 cc. of epinephrine intramuscularly within five minutes.[18] Cosgriff noted four patients with febrile reactions following injection of highly purified heparin. He had two other patients who developed sneezing, rhinitis, and conjunctivitis a few minutes after receiving injections of heparin. These manifestations were relieved by pyribenzamine.[19] DaCunha reports hemorrhage beneath the left rectus sheath following heparinization of a pregnant patient, from which the patient expired at surgery.[20] In Sweden four instances of true anaphylactoid reactions have occurred following heparin injections.[15]

The exact metabolism or disposition of heparin is not known. It is thought to be changed by an enzyme heparinase.[21] It was originally noted that salmine, one of the protamine class of proteins, combined with heparin to form a substance devoid of anticoagulant activity.[22] It has been found that heparin combines with protamine in a constant proportion of 0.3 mg. of heparin to 1.0 mg. protamine, and in this proportion the anticoagulant properties of heparin disappear.[3] In one study of the neutralization of heparin with protamine, 50 mg. of heparin were injected intravenously into 10 patients, and 15 minutes later salmine (protamine) was injected intravenously in doses ranging from 15 to 50 mg. In the patients receiving 40 to 50 mg. salmine, the elevated coagulation times were returned to normal levels within five minutes. No toxic reactions were noted in any patient during or after the injections.[23]

In the control of bleeding due to heparin, the first step is to withdraw the drug. Transfusions of whole fresh blood may be given to replace blood loss and to stimulate blood coagulation. Two substances, toluidine blue and protamine sulfate, have a specific antagonism for the anticoagulant properties of heparin. Both of these substances promptly restore normal coagulation time, but this effect may be transitory. The injections may have to be repeated several times. Two mg. per kilogram of body weight administered intravenously in 250 to 500 cc. of normal saline seems to be adequate dosage of either drug.

The toxicity of toluidine blue is relatively unknown. Although it is strongly hemolytic in dogs, no toxicity has been observed in man. Protamine sulfate is a foreign protein. Intravenous administration has resulted in severe anaphylactoid reactions in dogs, but no such reactions have been noted in man.[11]

DICUMAROL: DISCOVERY, SITE AND MODE OF ACTION

In the decade of 1920-1930, hemorrhagic tendencies were noted in large numbers of cattle that had eaten spoiled sweet clover hay. This condition plagued agriculturists for many years. During these years the agriculturists attempted to find the anticoagulant factor involved. Link and Campbell, after five years of intensive research in this field, isolated and crystallized what today we know as coumarin or dicumarol.[24, 25, 26]

The mechanism of the action of dicumarol is obscure. Dicumarol impairs the clotting mechanism by reducing the prothrombin content of the blood. Apparently it does not destory prothrombin since it is inactive in vitro. There is evidence of an inhibition of the synthesis of prothrombin by the liver, possibly by an interference with the utilization of vitamin K. Quick believes that prothrombin consists of an unstable component A linked by calcium with a stable component B, and that it is the latter stable component which is decreased by dicumarol. MacMillan has presented evidence suggesting the decrease in prothrombin produced by dicumarol is only apparent and that it is the prothrombin accelerator which is deficient. Dicumarol has been reported to inhibit platelet adhesiveness, which may be an additional beneficial action in the prevention of thrombosis.[52, 53]

DOSAGES AND METHODS OF ADMINISTRATION

Dicumarol is effective when administered orally, which is an advantage from the standpoint of simplicity of administration but a disadvantage from the standpoint of accuracy of dosage. The effect of dicumarol is delayed for 12 to 72 hours after a dose has been given, which is a disadvantage when rapid anticoagulant effect is desired. The effect of dicumarol may persist for 72 hours or longer once it has developed. While this is an advantage in maintaining the effect, it is a grave disadvantage when it is desired to stop the anticoagulant effect. The prolongation of prothrombin time varies greatly among different persons after equivalent doses of dicumarol, and the degree of response is usually unpredictable.[27] This variable response to dicumarol makes the control of its effects difficult to maintain.[28]

A good schedule to follow on any patient who is to receive dicumarol is to make a prothrombin determination, then give 300 mg. orally. Guide further usage by daily prothrombin determinations. When the prothrombin time is above 20 per cent of normal, 100 to 200 mg. of dicumarol may be given. If the prothrombin is less than 20 per cent of normal, dicumarol should be withheld.[27, 29, 30]

These physiological facts must be kept in mind in developing a rational schema for dicumarol dosage:

1. The drug may be absorbed slowly from the gastrointestinal tract. In some instances, peak plasma concentration following single oral doses may not be achieved for more than 24 hours.

2. Absorption may be incomplete. From zero to more than 40 per cent of a single oral dose may be eliminated in the stool.

3. The drug is not excreted in the urine.

4. The drug is metabolized in the body at a slow

A NEW BROAD SPECTRUM ANTIBIOTIC

"well tolerated by all age groups"



ACHROMYCIN, a new broad spectrum antibiotic, has proved its effectiveness in clinical trials among all age groups, and has definitely fewer side reactions associated with its use.

ACHROMYCIN maintains effective potency for a full 24 hours in solution, and provides rapid diffusion in tissues and body fluids.

ACHROMYCIN is effective against beta hemolytic streptococcic infections, *E. coli* infections, meningococcic, staphylococcic, pneumococcic and gonococcic infections, acute bronchitis and bronchiolitis, and certain mixed infections.

CAPSULES	250 mg. 100 mg. 50 mg.	SPERSOIDS* Dispersible Powder	50 mg. per teaspoonful (3.0 Gm.)	INTRAVENOUS	500 mg. 250 mg. 100 mg.

LEDERLE LABORATORIES DIVISION AMERICAN Cyanamid COMPANY PEARL RIVER, N. Y.

Lederle

*Reg. U.S. Pat. Off.

rate, and this varies considerably with the individual and the dose. Larger doses are transformed at a slower rate. Plasma levels may fall as slowly as 15 per cent per day or more rapidly than 50 per cent. Subjects in a stable metabolic state tend to degrade the drug in a more or less consistent manner.

5. The drug is widely distributed in most tissues. Repeating a dose while there is still dicumarol in the body results in an increment of a plasma level greater than the peak level achieved by that dose initially.

6. A threshold concentration of dicumarol in the plasma must be achieved before any prothrombin response can be detected. This level varies considerably in different subjects.

7. There is considerable individual variation in the "sensitivity" of the prothrombin mechanism to a given level of dicumarol. A level which results in a dangerously high prothrombin time in one subject may fail to induce a prothrombin response in another.

8. The pattern of the prothrombin response is roughly parallel to, and one to two days behind, the plasma dicumarol concentration. In some sensitive individuals the prothrombin time may remain elevated long after dicumarol has disappeared from the plasma.[31]

Factors to be considered in judging sizes of dosages of dicumarol are:

1. Weight of patient, since clinically the most accurate means discovered to date is on the basis of mgms. of dicumarol per kilogram of body weight.

2. Age is important due to greater frequency of hypersensitiveness found in elderly persons.

3. The nutritional state of the patient must be considered because increased sensitivity is noted frequently in those persons with poor nutrition.

4. Patients with severe anemia are often found to be hypersensitive to dicumarol.

5. Patients with cirrhosis are also frequently hypersensitive reactors.

6. The initial prothrombin time is important since larger doses are required in direct proportion usually to longer prothrombin times.

7. The urgency of therapy is important since, if the necessity is great and immediate, less attention is paid to the other factors.

8. If the patients have a past history of ulcerative gastrointestinal lesion or of any bleeding lesions, they must be carefully evaluated.[31]

COUMARIN DERIVATIVES

As the usage of dicumarol has become more widespread, attempts have been made to discover chemically similar substances that have about the same action but are superior. One of these is ethyl biscoumacetate, which is a synthetic coumarin derivative having somewhat similar chemical structure and anticoagulant action as dicumarol. This newer drug has been described under the various terms tromexan, pelentan, B. O. E. A., and D. E. A. While the anticoagulant effect is similar to that of dicumarol, the effect develops and disappears more rapidly than that of dicumarol, probably due to its greater solubility, more rapid absorption, and excretion through the urine. Ethyl biscoumacetate is approximately 1/5 as potent as dicumarol on the basis of mgm. per kilogram body weight. Therefore the dosage is five times that of dicumarol.[32, 33]

Another newer preparation is cyclocumarol which is related chemically and therapeutically to bishydroxycoumarin. This preparation is approximately two to three times as potent, usually more rapid and prolonged in its action than bishydroxycoumarin. Cyclocumarol is effective orally, and the same precautions must be taken as with other coumarin preparations.[34]

CONTRAINDICATIONS AND TOXICITY

As with most drugs, there are certain contraindications and dangers to the use of dicumarol. The contraindications are: (1) ascorbic acid and vitamin K deficiencies or hepatic disease, because anticoagulants may magnify the tendency to bleed; (2) renal insufficiency, because the effect of dicumarol is enhanced and prolonged in such cases; (3) blood dyscrasias in which there is impairment of the normal mechanisms for the prevention of bleeding; (4) recent surgical operations on the brain or spinal cord; (5) ulcerative lesions or open wounds, because of magnification of the tendency to bleed.

Frequently in the literature it is stated that the sole danger associated with the use of dicumarol is hemorrhage.[35] Allen et al. in a study of 1,938 postoperative cases in which dicumarol was used, discovered minor hemorrhages occurred in 3.4 per cent of cases and serious bleeding occurred in 1.8 per cent of cases. Two cases of hemorrhage resulted in fatalities.[35] Gripe reported 3 cases of serious hemorrhage in 125 patients given tromexan. One of these three expired following gross hematuria and subarachnoid hemorrhage.[36] Lilly and Lee reported five deaths due to dicumarol therapy.[37] Wright and Rothman reported four deaths from dicumarol therapy; all four patients had prothrombin levels in the therapeutic range, 25 to 40 per cent of normal.[38] Powers reported two patients who developed severe hemorrhage as a result of dicumarol therapy. One of these patients died and was found to have pulmonary and retroperitoneal hemorrhages.[39] Cave reported one patient who developed hemorrhagic diatheses and hematuria on dicumarol therapy.[40] One case is found in the literature of a nurse who administered dicumarol in an undetermined amount to herself with resultant hematuria and hemorrhagic diatheses.[41] Crane reported a case of massive hematuria following administration of 2,300 mg. of dicumarol in a period of

Earlier Blood Levels *from*

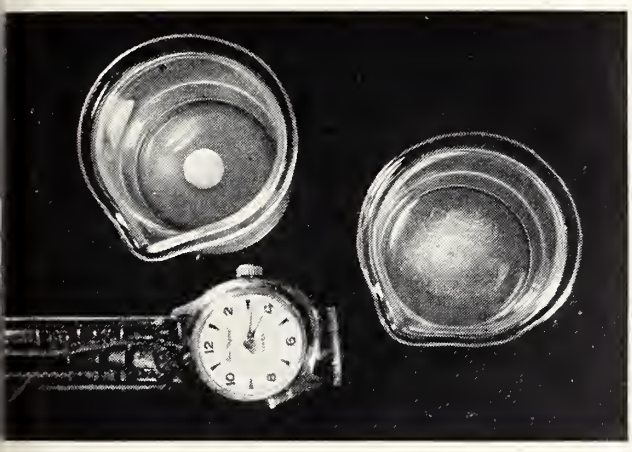
FILM SEALED*

ERYTHROCIN

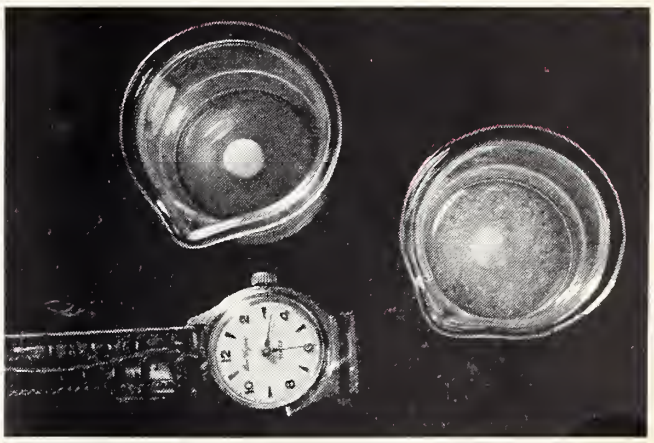
TRADE MARK

DISINTEGRATES FASTER THAN ENTERIC COATING

HIGH BLOOD CONCENTRATIONS WITHIN 2 HOURS



3:20—Five minutes later, *Film Sealed* coating has already started to disintegrate. The tissue-thin film actually begins to dissolve within 30 seconds after patient swallows tablet.



3:30—*Film Sealing* is now completely dissolved. At this stage, ERYTHROCIN is ready to be absorbed, and ready to destroy sensitive cocci—even those resistant to most other antibiotics.



3:45—Now the *Film Sealed* tablet mushrooms out with all of the drug available for absorption. Note that enteric-coated tablet is still intact. Tests show that the new Stearate form definitely protects ERYTHROCIN against gastric acids.



4:00—Because of *Film Sealing* (marketed only by Abbott) the drug is released faster, absorbed sooner. In the body, effective ERYTHROCIN blood levels now appear *in less than 2 hours* (instead of 4-6 hours as before).

Abbott

*pat. applied for

406150

seven days.[42] One case of a hemorrhage beneath the right rectus sheath of a dicumarolized patient is reported.[43] Boller et al. reported 85 patients treated with dicumarol with three deaths from pulmonary embolism.[44]

TREATMENT OF OVERDOSAGE

In past literature, synthetic vitamin K has been suggested as the treatment for dicumarol overdosage. Water soluble vitamins have the advantage of ease of administration. However, in recent literature, their effectiveness has been doubted and considered little more valuable than the simple withdrawal of dicumarol.

The hypoprothrombinemia induced with dicumarol is much more difficult to control than that which is found in other states characterized by deficient intake or absorption of vitamin K.

One study compared synkayvite, Menadione sodium bisulfite, and vitamin K_1 oxide on their effectiveness for counteracting the hypoprothrombinemia of dicumarol. It was found that emulsions of vitamin K_1 oxide administered intravenously in doses of 500 to 2000 mg. were strikingly superior to the other substances tested. The therapeutic goal was attained in approximately 1/10 the time required for synkayvite, menadione sodium bisulfite, or the normal controls. Of 26 patients treated with vitamin K_1 oxide, only one required more than one day to achieve the therapeutic result.[45] With a single oral dose of 500 mg. of vitamin K_1, the prothrombin time was reduced to less than 27 seconds (30 per cent prothrombin activity) in 15 of 16 dicumarolized patients in less than 24 hours.[46]

The intravenous administration of the vitamin K_1 preparations has been difficult due to their oil solubility. However, now there have been prepared special emulsions that can be administered intravenously.[47]

In patients given tromexan until prothrombin levels of 10-30 per cent were obtained, it was found that prothrombin activity was out of the dangerous range within 16 hours after administration of 5 mg. per kilogram body weight of purified K_1 in an emulsified form.[48]

In 7 patients with dicumarol-induced hypoprothrombinemia that were given intravenous emulsions of vitamin K_1, all showed a prompt decline in prothrombin time. When a dose in the range of 10 to 20 mg. per kilogram was administered, the prothrombin times of all the patients fell to 36 seconds or below in 120 minutes and to 30 seconds or below in 5 hours.[49] In a recent study using synthetic vitamin K to neutralize hypoprothrombinemia induced by dicumarol, it was found that only about 25 per cent of the patients studied had prothrombin times of 30 seconds or under within 24 hours.[50] Shapiro and his group state that recent literature regarding the uselessness of synthetic vitamin K is a result of the use of ineffective dosages of these drugs.[51]

The general consensus is that water soluble, synthetic vitamin K preparations have been used and reversals of hypoprothrombinemia have been demonstrated. However, it has been demonstrated that emulsions of oil soluble vitamin K_1 and vitamin K_1 oxide are far superior to the water soluble vitamin K in effectiveness per mg., rapidity of action, and duration of effect.

SUMMARY

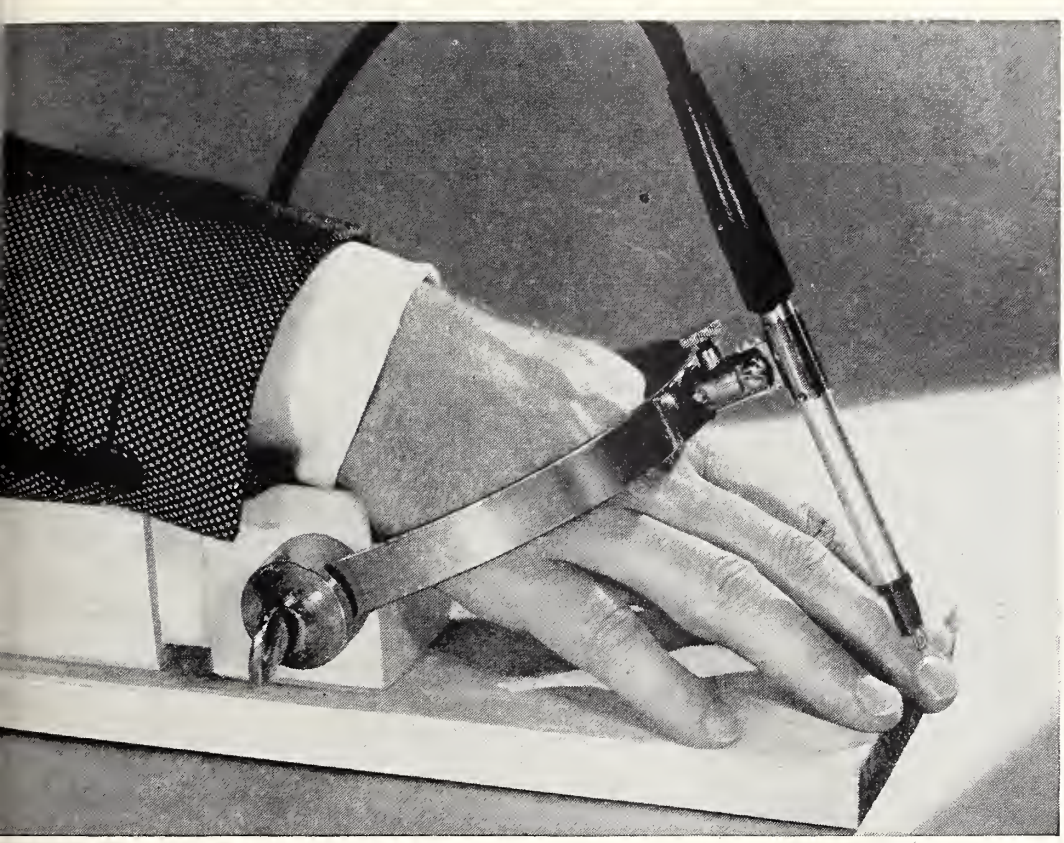
Heparin as an anticoagulant acts to inhibit formation of thrombin and fibrin. It also inhibits agglutination of platelets. Heparin cannot be given orally, but it may be given intravenously or intramuscularly in the newer, slow acting preparations. Heparin has a rapid action with the anticoagulant effect usually being evident in a matter of minutes. The anticoagulant effect of heparin is ascertained by the Lee and White clotting time with the usual therapeutic level at least three times the normal level. Some instances of anaphylactoid reactions have followed administration of heparin, but the greatest danger lies in bleeding, which may be controlled by administration of protamine sulfate.

Dicumarol, given orally, acts to reduce the prothrombin content of blood. Dicumarol is slow in its effect, requiring 12 to 72 hours, and the effect is often prolonged to 72 hours after cessation of the medication. Therapeutic effect is followed by prothrombin times with a level slightly above the minimum of 20 per cent of normal desired. The chief danger of overdosage of dicumarol is hemorrhage, which may be controlled by administration of synthetic water soluble vitamin K or emulsions of vitamin K_1, with the latter being the drug of choice. Due to the variation in time of action of the two drugs, they are frequently used at the same time until the prothrombin time is within the therapeutic range, at which time the heparin is discontinued.

BIBLIOGRAPHY

1. Lucia, S. P.: Use of anticoagulants, heparin and dicumarol, California Medicine 65:5-8 (July) 1946.
2. Mason, M. F.: Heparin, Surgery 5:451, 1939.
3. D'Alessandro, A. J.: Heparin; its properties and clinical use, Surgery, Gynecology and Obstetrics (Internat'l. Abstr. Surg.) 74:62, 1942.
4. Holyoke, J. B., and Wheelwright, H. J.: The effect of Depo-Heparin on the prothrombin time, New England Journal of Medicine 246:1 (Jan. 3) 1952.
5. Babb, Audrie Leah: The blood clotting mechanism, Jour. of the American Med. Women's Association 2:4, 166-168 (April) 1947.
6. MacLean, J.: The thromboplastic action of cephalin, Am. J. Physiol. 41: 250, 1916.
7. Smith, Ralph G.: The pharmacology of anticoagulant agents, New Orleans Med. and Surg. Journal 102:4 (Oct.) 1949.
8. Holder, Cole, and Davis: Clinical studies of heparin cofactor, Surgery, Gyn. and Obstetrics, 89:20-23 (July) 1949.
9. Nichol, E. Sterling: Clinical use of heparin and dicumarol, New Orleans Med. and Surg. Journal 102:5 (November) 1949.
10. Barker, Nelson W.: Anticoagulant therapy, Journal of the Omaha Mid-West Clinical Soc. 9:3 (August) 1948.
11. Marple, Charles: The administration of anticoagulants, California Medicine 73:166 (August) 1950.
12. Stats, Daniel, and Neuhof, Harold: Concentrated aqueous heparin, Am. Jour. of Med. Sc. 214:159-162, 1947.
13. Smiles, William: Long acting heparin preparation, U. S. Armed Forces Med. Jour. II:45 (January) 1951.
14. Wynn, Goodwin, and Birbeck: Prolonged anticoagulant

Physiological test compares Kent's "Micronite" Filter with other cigarette filters



"KENT" AND "MICRONITE" ARE REGISTERED TRADEMARKS OF P. LORILLARD COMPANY

To compare the efficiency of various filters as they affect physiological responses in the cigarette smoker, drop in surface skin temperature at the last phalanx was measured.

Using well-established procedures, the subject smoked conventional filter cigarettes and the new KENT with the exclusive Micronite Filter.

For every other filter cigarette, the drop in temperature averaged over 6 degrees. For KENT's Micronite Filter, there was no appreciable drop.

These findings confirm the results of other scientific measurements that show these facts: 1) KENT's Micronite Filter takes out *far more* nicotine and tars than any other cigarette, *old or new*. 2) Ordinary cotton, cellulose or crepe paper filters remove a small but ineffective amount of nicotine and tars.

Thus KENT, with the first filter that really works, gives the one smoker out of every three who is susceptible to nicotine and tars the protection he needs . . . while offering the satisfaction he expects of fine tobacco.

For these reasons, smokers have made the new KENT the most popular new brand of cigarette to be introduced in the last 20 years.

If you have yet to try the new KENT with the exclusive Micronite Filter, may we suggest you do so soon?



therapy with heparin, British Med. J. 1:4764, 893-896 (April 26) 1952.
15. Duff, Birch: The administration of heparin, Surg., Gyn. and Obst. 93:3 (Sept.) 1951.
16. DeTakats, G.: Thrombo-embolism, J. Internat. Chir. 8:903, 1948.
17. Gotz, Alexander: Severe spontaneous hypersensitivity to heparin, Annals of Internal Med. 35:4, 912-922 (October) 1951.
18. Chernoff, Amoz I.: Anaphylactic reaction following injection of heparin, New England Journal of Med. 242:315, 1950.
19. Cosguff, Cross, and Habif: The management of venous thrombosis and pulmonary embolism, Surg. Clinics N. America 28:324-338, 1948.
20. DaCunha: Massive haemorrhage following anticoagulant therapy in pregnancy, J. of Obst. and Gyn. of the Brit. Emp. 59:4, 521-522 (August) 1952.
21. Pfeiffer and Sain: Heparin and dicumarol, Surg., Gyn. & Obst. (Internat'l. Abstr. Surg.) 78:109, 1944.
22. Heathcote, Reginald: The clotting of the blood and anticoagulants, The Practitioner 160, 1948.
23. Parkin and Kvale: Neutralization of heparin with protamine (saline), J. Lab. and Clin. Med. 32:1396 (November) 1947.
24. Link, K. P.: The anticoagulant from spoiled sweet clover hay, The Harvey Lectures 39:162-216, 1944.
25. Link, K. P.: The anticoagulant 3, 3'-methylanebis, Federation Proceedings 4:176-182 (June) 1945.
26. Link and Campbell: Studies on hemorrhagic sweet clover disease, J. Biol. Chem. 138:21-33, 1941.
27. Barker, Hines, Kvale, and Allen: Dicumarol, Am. Jour. of Med. 3:634-642 (November) 1947.
28. Burt, C. C.: Anticoagulants, Edinburgh Med. J. 56:632-640 (Nov.-Dec.) 1947.
29. Coleman, F. C.: The prothrombin time in dicumarol therapy, Annals of Internal Med. 30:895 (May) 1949.
30. Levan, John B.: Dicoumarol therapy, Ann. Internal Med. 25:941 (December) 1946.
31. Shapiro, Shepard, and Weiner, Murray: An evaluation of methods of dicumarol administration, American Heart Jour. 41:749-754 (May) 1951.
32. Barker, Estes, and Mann: Clinical experiences with the anticoagulant ethyl biscoumacetate, Proc. Staff Meet. Mayo Clinic 26:162-167 (April 25) 1951.
33. Hausner, Shafer, Corson, Johnson, Trujillo, and Langham: Clinical evaluation of dicumarol derivitives with a metabolic study of radioactively labeled anticoagulants in animals, Circulation III: 2 (February) 1951.
34. New and Nonofficial Remedies: Cyclocumarol, J.A.M.A. 148:11 (March 15) 1952.
35. Allen, E. V.; Hines, E. A.; Kvala, W. F., and Barker, N. W.: The use of dicumarol as an anticoagulant, Ann. Internal Med. 27:371 (September) 1947.
36. Gripe, Richard P.: Severe hemorrhage due to tromeian, New England Journal of Medicine 245:21 (November 22) 1951.
37. Lilly, G. D., and Lee, R. H.: Complications of anticoagulant therapy, Surgery 26:957, 1949.
38. Wright, Louis, and Rothman, Milton: Deaths from dicumarol, A.M.A. Archives of Surg. 62:23-28 (January) 1951.
39. Powers, John L.: Toxicity of dicumarol, Ann. Int. Med. 32:146-152 (January) 1950.
40. Cave, H. A.: Haemorrhagic manifestations secondary to dicumarol, Canadian M.A.J. 61:520-523 (November) 1949.
41. Draper, Arthur: Dicumarol poisoning, J.A.M.A. 136:171-172 (January) 1948.
42. Rosenbloom, David, and Crane, Jay J.: Massive hematuria due to dicumarol overdosage, J.A.M.A. 132:924-925 (December 14) 1946.
43. Miles, Kane, Degenshein: A complication of anticoagulant therapy simulating acute surgical abdomen, New York State Jour. of Med. 51:1197 (May) 1951.
44. Boller, Quigley, Tarpey, and Harris: Experiences with anticoagulant therapy, New York State Jour. of Med. 49:14 (July 15) 1949.
45. James, Bennett, Scheinberg, and Butler: Clinical studies on dicumarol hypoprothrombinemia and vitamin K preparations, Archives of Internal Med. 83:6 (June) 1949.
46. Fuller, Benjamin, and Barker, Nelson: The effect of vitamin K_1 administered orally on the control of the coagulation defect induced by dicumarol, Minnesota Med. 34:326-328 (April) 1951.
47. Shoslakey, Geyer, Ye, and Stare: The treatment of dicumarol induced hypoprothrombinemia in dogs witb emulsified vit. K_1 administered intravenously, Journal of Lab. and Clinical Med. 36:531 (October) 1950.
48. VanBuskirk, William: The effect of intravenous injection of emulsified vitamin K_1 on the hypoprothrombinemia induced by tromexan, New England Journal of Medicine 248:57-58 (January 8) 1953.
49. Watkin, Van Itallia, Logan, Geyer, Davidson, and Stare: The treatment of dicumarol induced hypoprothrombinemia in man with emulsified vitamin K_1 administered intravenously, J. Lab. and Clin. Med. 37:269-271 (February) 1951.
50. Overman, Sorenson, and Wright: Effectiveness of synthetic water soluble vitamin K preparations in bishydroxycoumarin induced hypoprothrombinemia, J.A.M.A. 145:393-399 (February 10) 1951.
51. Shapiro, Weiner, and Simson: The effect of water soluble preparation of vitamin K in dicumarol induced hypoprothrominemia, New England Journal of Medicine 243:775-779 (November 16) 1950.
52. Smith, R. G.: Pharmacology of anticoagulant agents, New Orleans M. and S. J. 102:159-162 (October) 1949.
53. MacMillan, Robert: Observation on the mechanism of action of dicoumarol, Science 108:416-417 (October 15) 1948.



PEDIGREED IN ITS FIELD

Audivox, successor to Western Electric Hearing Aid Division, brings the boon of better hearing to thousands.

These are the Audivox Hearing Aid Dealers who serve you in Kansas. Audivox dealers are chosen for their competence and their interest in your patients' hearing problems.

THE TOPEKA HEARING CENTER
106 West 8th Street
Topeka, Kansas
Tel: 4-9609

KANSAS HEARING CENTER
223 Broadway
Wichita, Kansas
Tel: 4-8614

PROFESSIONAL HEARING SERVICE
1616 South 21st Street
Lincoln, Nebraska

ROSS HEARING CENTER
P. O. Box 528
Elm Creek, Nebraska

O. E. KULLBERG
110 Forest Boulevard
Hastings, Nebraska

SOUTHWESTERN HEARING AID COMPANY
202 12th & Walnut Building
Kansas City, Missouri
Tel: Harrison 0541

SOUTHWESTERN HEARING AID COMPANY
216 North Seventh Street
St. Joseph, Missouri
Tel: 4-5064

audivox
Successor to Western Electric Hearing Aid Division



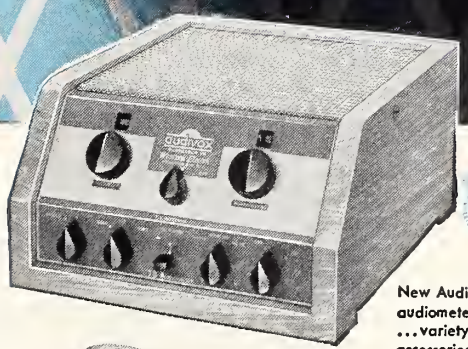
blueblood

Only a long and distinguished ancestry of champions can produce a feline blueblood.

Only **audivox** in the hearing aid field can trace an ancestry that includes both Western Electric and Bell Telephone Laboratories. **audivox** lineage springs from the pioneer experiments of Dr. Alexander Graham Bell, which were furthered by the development of the hearing aid at Bell Telephone Laboratories, and in turn, brought to fruition by Western Electric and **audivox** engineers.

Distinctly a blueblood in its field, **audivox**, successor to Western Electric Hearing Aid Division, brings the boon of better hearing, and its enrichment of living, to thousands. With the magical modern transistor, with scientific hearing measurement and scientific instrument-fitting, serviced by a nationwide network of professionally-skilled dealers, **audivox** moves forward today in a proud tradition.

TO THE DOCTOR: *If you use or need an audiometer there is in every major city from coast to coast a career Audivox dealer, chosen for his integrity and ability, who will be glad to show you why an Audivox audiometer will serve you best.*



New Audivox audiometer 7BD ...variety of accessories available



Alexander Graham Bell

audivox

Successor to Western Electric Hearing Aid Division

123 Worcester, St., Boston, Mass.

THE MONTH IN WASHINGTON

At the request of the Defense Department, Congress is considering a bill to expand and make more uniform the medical care program for civilian dependents of military personnel. It could have significant impact on the practice of medicine and on medical economics.

The legislation developed out of the Defense Department's Moulton Commission report of a year ago. In the intervening months the department's legislative planners called in representatives of the American Medical Association and other professional groups for advice. But the bill finally presented to Congress is evidence that not all differences of opinion were compromised. While in many respects the measure is in line with the policy of the A.M.A. on dependent care, at least one basic conflict remains:

The department's bill states that dependents should receive private medical care only when military facilities are unavailable or inadequate. The A.M.A.'s policy, adopted after long study of the problem, is that dependents should be cared for in military hospitals and by uniformed physicians only when civilian care is inadequate or unavailable.

There is almost complete agreement that the present patchwork dependent medical care program should be changed to make benefits uniform geographically and within the services, and to spell out the benefits in law. The issue is whether the military medical services should care for all qualified civilian dependents, or dependents should, like the rest of the population, get their medical care from civilian physicians and hospitals.

Under the bill, medical care furnished by or underwritten by the federal government would be limited to "diagnosis, acute medical and surgical conditions, contagious diseases, immunization, and maternity and infant care." Dental care would be allowed only in emergencies or as an adjunct to medical care. These restrictions would be waived overseas and at remote stations in the United States.

The definition of "dependents" would not extend beyond parents and parents-in-law, and these relatives would have to receive at least half their support from the military member to qualify.

The Secretary of Defense would decide what charges, if any, to levy against dependents treated at military facilities. When treated privately, the dependents would pay the first $10 cost of any illness, plus not more than 10 per cent of the total cost. The secretary could make use of voluntary health insurance for dependents if this system were found to be more economical.

The Senate Armed Services Committee was slow to take up the dependent care bill because of a heavy schedule of other hearings. Nor did it make fast progress in the House. There the introduction of the bill was delayed when Chairman Dewey Short (R., Mo.) called on the Defense Department to furnish him with detailed information on what the new medical care program would cost.

By mid-May, when Congress had about concluded hearings on all major administration health bills, a new factor was introduced. Chairman Wolverton of the House Interstate and Foreign Commerce Committee called hearings on his own bill for federal guarantee of private loans to health facilities. This was not part of the original Eisenhower health program, but there were some indications that the administration might get behind it.

As originally drawn, the bill would virtually exclude all clinics and hospitals except those operated in conjunction with prepaid insurance plans. During the hearings, Mr. Wolverton indicated he would be willing to drop this restriction. If this were done, the law then would offer benefits to all—fee-for-service physicians and groups as well as "closed panels."

During this period, some sentiment developed to combine the loan guarantee bill with the reinsurance bill, which wasn't making much progress on its own. The result was a period of confusion and uncertainty, with no clear indication of what either the committee or the administration really wanted.

A few other medically-important bills were advancing on schedule. The House Ways and Means Committee gave every indication of reporting out a bill to require all employers (physicians included) to participate in the federal-state unemployment insurance program. As usual moving faster than the Senate, the House had passed a bill to give state health officers more control over federal grants for public health work. The House also was nearing a vote on extension of the social security program, with no suggestion that physicians and other self-employed groups who don't want coverage would be exempted. The House-approved Hill-Burton expansion bill was waiting action in the Senate.

CLASSIFIED ADVERTISEMENTS

FOR SALE—X-ray equipment; complete Profexray, 100 MA 100 KVP, double tube unit. None better made, used very little, practically new, save $1,000. Write the Journal 11-54.

WANTED: Dermatologist for seven-man group in Midwestern city. Above average facilities with excellent financial opportunity. Write the Journal 13-54.

PHYSICIAN leaving in August or before for one-year residency wants replacement. Small community. Home and office available. Office equipped with EKG, x-ray, all other necessities. Write the JOURNAL 8-54.

LOCATION—Town of 9,000, 2 colleges, hospital, not overcrowded with doctors, all swell fellows. Modern ground floor office, not much to sell. Partnership if desired. Give qualifications and personal data. Write the JOURNAL 10-54.

WANTED: Pediatrician for seven-man group in Midwestern city. Above average facilities with excellent financial opportunity. Write the Journal 12-54.

PATRONIZE JOURNAL ADVERTISERS

It is advertisers who make possible the publication of the Journal in its present form.

They are deserving of your patronage.

RENTALS – SALES

HOME PATIENT AIDS
- Bed Pans
- Urinals
- Crutches
- Wheel Chairs
- Invalid Walkers

SICK ROOM EQUIPMENT
- Hospital Beds Complete
- Bedside Commodes
- Bedside Tables
- Invalid Lifts
- Heat Lamps

Many Other Items

★ **PETRO'S RENTAL SERVICE** ★

618-20 Quincy — Topeka, Kansas — Phone 40207

Pure as sunlight



Attendance at Cancer Conference

A recent relase from the American Cancer Society, Kansas Division, compares physician attendance at its Sixth Annual Mid-West Cancer Conference with attendance at those held in previous years. The following figures were given: 1949, 126; 1950, 198; 1951, 208; 1952, 261; 1953, 235; 1954, 279. In addition to the 279 physicians who were present in 1954, there were also registrations for 17 interns and nurses, 7 guests, and 24 speakers and other out-of-state physicians, a total of 327.

The states represented, in addition to Kansas, were: California, Colorado, Illinois, Massachusetts, Missouri, Nebraska, New York, Oklahoma, and Pennsylvania. There was also one registrant from Greece and one from Korea.

As a group, older students are more likely to drop out without finishing medical school than are their younger companions, according to a report in a recent issue of *Journal of Medical Education*. Possible reasons listed include the fact that older students may not be as healthy as younger ones, they may have more financial burdens, they have more time-consuming social obligations, they may lack real motivation, and they may find adjustment difficult.

Everything for the Laboratory

SOUTHWEST SCIENTIFIC CORPORATION

LABORATORY SUPPLIES AND EQUIPMENT

122 South St. Francis Street
Phone 2-0582 Wichita, Kansas

Nationally advertised Surgical Supplies and Equipment have been placed at Topeka, Joplin, Kansas City and St. Joseph for your convenience by—

GOETZE NIEMER COMPANY

Management by Dr. W. F. Goetze, member of the American Medical Ass'n, assures intelligent servicing of your orders.

It's Convenient to Buy Kanox

THERAPEUTIC OXYGEN AND ANESTHETIC GASES

Oxygen Therapy
Regulators and Equipment
Repair Service for
Regulators, Flowmeters
and Humidifiers



Nitrous Oxide
Ethylene
Carbon Dioxide
Helium
Oxygen
Cyclopropane

KANSAS OXYGEN, Inc.

Phone 5-5551 HUTCHINSON, KANSAS Box 551

THE LATTIMORE-FINK LABORATORIES

Topeka — El Dorado
Kansas

J. L. Lattimore, A.B., M.D., Pathologist
A. A. Fink, A.B., M.D., Pathologist
H. C. Ebendorf, M.T., Serologist
A. C. Keith, B.S., Chemist
L. W. Hull, A.B., Bacteriologist
Walter Norris, A.B., Chemist

Anatomical and Clinical Pathology

Containers Furnished Upon Request

MUTUAL TRUST

Offered by

MUTUAL DISTRIBUTORS, Inc.
1016 Baltimore • Kansas City 5, Mo.

Principal Underwriter and Distributor

PROSPECTUS ON REQUEST

PLEASE SEND ME PROSPECTUS

*Name*_____

*Address*_____

*City*_____ *State*_____

THE MEDICAL PROTECTIVE COMPANY
FORT WAYNE, INDIANA

PROFESSIONAL PROTECTION EXCLUSIVELY SINCE 1899

specialized service assures "*know-how*"

TOPEKA Office:
J. E. McCurdy, Rep.,
1035 Randolph Avenue,
Telephone 2-3027

Deformity Appliances of Quality

Orthopedic and Surgical Appliances

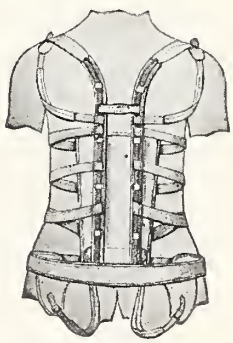
Artificial Limbs
Trusses

Abdominal Supports

Elastic Hosiery

Foot Supports

Surgical Corsets



Taylor Back Brace
Made to Order in
Our Own Factory

P. W. HANICKE MFG. CO.
1009 McGee St. Victor 4750
KANSAS CITY, MO.

DEATH NOTICES

Mowry Stafford Thacher, M.D.

Dr. M. S. Thacher, 84, Turon, who was honored at a community celebration last year in recognition of 50 years of service to the city, died at his home on April 1. He was an honorary member of the Reno County Society. Dr. Thacher received his medical degree from the Kansas Medical College, Topeka, in 1902 and began his practice in Turon. Except for two years spent in Blue Rapids, he continued practice in Turon until his death.

Edward D. Kilbourn, M.D.

Dr. E. D. Kilbourn, 73, Wichita obstetrician and gynecologist, died on April 17 in Rochester, Minnesota, following surgery. He had been an active member of the Sedgwick County Society for 49 years. A graduate of Northwestern University Medical School in 1904, Dr. Kilbourn took postgraduate work at the University of Vienna, Austria, before beginning practice.

Orlan Dayton Sharpe, M.D.

Dr. O. D. Sharpe, 85, an honorary member of the Wilson County Society and a past president of that group, died at his home at Neodesha on May 1. He was graduated from Kansas City Medical College in 1895 and received his Kansas license in 1901. He had practiced in Neodesha from that time until his retirement in 1946. On the occasion of his 50th anniversary in the practice of medicine, he was honored by the county society at a dinner.

Leadership is that quality that lifts men and women toward whom one has responsibility out of their ordinary selves and permits them to accomplish in their fields more than they could if the leader were not there to impose his personality upon them. Of all the qualities which go into leadership, probably the most important is the capacity to command instinctive respect. But in order to command instinctive respect, one must have qualities of integrity, self-confidence, and acumen.—*Frank Pace, Jr.*



Woodcroft Hospital--Pueblo, Colorado

A private hospital for the scientific treatment of neuro-psychiatric disorders, including alcoholism and drug addiction. Beautiful landscaping and home-like surroundings afford a restful atmosphere. Accommodations vary from single rooms with or without bath to rooms en suite, allowing for segregation of guests.

Detailed information furnished on request.

KARL J. WAGGENER, M.D.

Given social acceptance,

the great majority of

epileptic patients

can lead normal lives

DILANTIN, after more than 15 years of clinical experience, is an established anticonvulsant of choice. Its ability to control grand mal and psychomotor seizures, without the handicap of hypnosis, helps many epileptic individuals participate in normal educational, economic, and social activities.

Dilantin® Sodium

(diphenylhydantoin sodium, Parke-Davis)

DILANTIN Sodium is supplied in a variety of forms—including Kapseals® of 0.03 Gm. (½ gr.) and 0.1 Gm. (1½ gr.) in bottles of 100 and 1000.

MEDICAMENTA VERA · P Co.

Parke, Davis & Company

DETROIT, MICHIGAN

TABLE OF CONTENTS

JULY, 1954

ORIGINAL ARTICLES

Carcinoma of the Cervix—L. A. Calkins, M.D., Kansas City, Kansas 377

Spontaneous Intravascular Clotting—William W. Abrams, M.D., Kansas City, Kansas 379

Poisonings with the Newer Pesticide Compounds—Thomas R. Hood, M.D., and Evan Wright, Topeka, Kansas 384

The Doctor Goes to Court—Joseph Cohen, LL.B., Kansas City, Kansas 388

EDITORIALS

Federal Health Programs 395

Malpractice Insurance 396

Physician Placement 398

MISCELLANEOUS

President's Page 394

Clinicopathological Conference 408

The Complications of Polycythemia Vera—Senior Thesis 399

Entered as second-class matter at the post office at Fulton, Missouri, June 19, 1953, under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of October 3, 1917, authorized on July 2, 1918. Office of Publication: 1201-05 Bluff Street, Fulton, Missouri.

THE JOURNAL OF THE KANSAS MEDICAL SOCIETY

Editorial Board

ORVILLE R. CLARK, M.D., *Editor,* Topeka

JOHN W. CAVANAUGH, M.D., Topeka

DAVID E. GRAY, M.D., Topeka

RICHARD GREER, M.D., Topeka

DWIGHT LAWSON, M.D., Topeka

Associate Editors

DONALD P. TREES, M.D., Wichita

GLEN R. SHEPHERD, M.D., Kansas City

Information

Ownership: The Journal is a non-profit publication owned and publisbed monthly by the Kansas Medical Society.

Subscription: A year's subscription to the Journal is included in membership in the Kansas Medical Society, with $2.00 of each member's dues apportioned to the Journal. Rates to others, except in foreign countries, $4.00 per year or 60c per copy.

Material: Scientific articles, editorials, and data of general interest are invited from all members. Articles are to be submitted on condition that they are contributed solely to this publication. A right is reserved to reject any material deemed unsatisfactory.

Manuscripts: Only manuscripts that are typewritten on one side, double spaced, and original copies can be accepted. Manuscripts will be returned upon request.

Advertising: All advertising contracts, and all copy from advertisers under contract are subject to approval of the editorial board. Copy sbould be received by the 20th of the month immediately preceding the month of publication.

Non-Responsibility: Although an effort is made to publish only accurate articles and legitimate advertisements. the Journal denies legal responsibility for any statements, opinions, or advertisements appearing under the names of contributors or concerns.

PAULINE FARRELL, Managing Editor and Advertising Manager

OLIVER E. EBEL, Business Manager

THE KANSAS MEDICAL SOCIETY

President . . . . . MURRAY C. EDDY, M.D., Hays

Past President . . . . . LUCIEN R. PYLE, M.D., Topeka

President-Elect . . . . . JOHN M. PORTER, M.D., Concordia

First Vice-President . . . CLYDE W. MILLER, M.D., Wichita

Second Vice-President, CONRAD M. BARNES, M.D., Seneca

Constitutional Secretary, JAMES A. BUTIN, M.D., Chanute

Treasurer . . . . . JOHN L. LATTIMORE, M.D., Topeka

A.M.A. Delegate, 1954-1955, GEORGE F. GSELL, M.D., Wichita

A.M.A. Delegate, 1955-1956, LAURENCE S. NELSON, M.D., Salina

Councilors and Councilor Districts

First District, FREDERICK E. WRIGHTMAN, M.D., Sabetha

Second District . . . GLENN R. PETERS, M.D., Kansas City

Third District . . . . . H. PENFIELD JONES, M.D., Lawrence

Fourth District . . . . CHARLES E. VESTLE, M.D., Humboldt

Fifth District, SEVERT A. ANDERSON, M.D., Clay Center

Sixth District . . . . . FLOYD C. TAGGART, M.D., Topeka

Seventh District . . . . . TO BE ANNOUNCED

Eighth District . . . . . JAMES E. HILL, M.D., Arkansas City

Ninth District . . . . LAURENCE S. NELSON, JR., M.D., Salina

Tenth District . . . . . HAROLD M. GLOVER, M.D., Newton

Eleventh District . . . . NORTON L. FRANCIS, M.D., Wichita

Twelfth District . . . . . CYRIL V. BLACK, M.D., Pratt

Thirteenth District . . LLOYD W. REYNOLDS, M.D., Hays

Fourteenth District . . . . JUSTIN A. BLOUNT, M.D., Larned

Fifteenth District . . . . ROBERT G. KLEIN, M.D., Dodge City

Sixteenth District . . . . . JAMES L. JENSON, M.D., Colby

Seventeenth District . . . . . H. PRESTON PALMER, M.D., Scott City

OLIVER E. EBEL, EXECUTIVE SECRETARY

RUEBEN M. DALBEC, EXECUTIVE ASSISTANT

Executive Office: 315 West Fourth Street, Topeka

THE JOURNAL
of the
KANSAS MEDICAL SOCIETY

Copyright, 1954, by The Kansas Medical Society

Volume LV JULY, 1954 No. 7

Carcinoma of the Cervix

L. A. Calkins, M.D.
Kansas City, Kansas

That the cure rate of carcinoma of the cervix has been materially improved in the last 20 years is well shown by Dr. Heyman[1] in his last report. In fact, more than twice as many women are being cured of this disease today as were cured 20 years ago. This improvement has come about in two ways. First, there have been larger amounts of radium made available in better and more applicable forms in treatment centers. Whether the new radioactive isotopes will further improve this situation remains to be seen. Voltage of x-ray therapy machines has been increased, and it is not yet apparent what the optimum voltage will prove to be. Quite certainly, the 250,000 volt machine has produced better results than the 150,000 volt machine formerly used.

The more recent studies of Dr. Franklin L. Payne,[2] Dr. Willis E. Brown,[3] and others give promise of further improvements in methods of radiation therapy. Recent experiments at surgical attack have been enlightening, but, so far, have failed to measure up to the results obtained with radiation therapy. In the small per cent (less than 10 per cent) of radiation failures, a proper field of surgical therapy investigation is being exploited in several places. So far, the results of this therapy, as far as ultimate cure is concerned, have been extremely disappointing. Moreover, the complications and injuries to the urinary and intestinal tracts have been no less than appalling. Whether the present extensive and expensive effort of Dr. Meigs to determine this radiation failure early and, therefore, to afford a better chance of surgical success, will prove fruitful, remains to be seen. Such a method, because of its expense, is prohibitive to most clinics.

Second, the American Cancer Society, by its educational campaign, has also contributed much in the way of getting earlier diagnosis and earlier therapy for such patients. As will be shown later, the cure rate of early carcinoma of the cervix is now 85 per cent to 90 per cent, approximately the cure rate of most benign lesions subjected to surgery. That we will ever achieve such an early diagnosis in all patients now seems utterly impossible. That we can completely avoid the too late and, therefore, far advanced tumor in some patients seems likewise impossible. That the far advanced cases should be seldom seen seems entirely possible.

Another feature of the avoidance of bad results has not been sufficiently emphasized. I refer to the so-called "lag period," which is the length of time elapsing between the first visit of the patient to her doctor and the application of definitive therapy. Twenty years ago, this lag period averaged 13 months. Part of this was due to the fear on the part of the patient that any therapy applied would serve to "spread" the disease rather than cure it. Another part was due to the more or less widespread feeling on the part of the general practitioner, and some specialists, that the situation was a more or less hopeless one, and his consequent failure to urge immediate treatment. Fortunately, this long period of 13 months delay has been reduced to about six months. That it should be further reduced is obvious.

Table I shows experience with those patients coming to the University of Kansas Hospital and treated

From the University of Kansas Medical Center.

TABLE I
CARCINOMA OF THE CERVIX
Incidence

	Stage I	*Stage II*	*Stage III*	*Stage IV*	*Totals*
1929-42	32 (7%)	113 (26%)	190 (44%)	100 (23%)	435
1942-48	44 (18%)	76 (31%)	88 (36%)	36 (15%)	244
1949-53	28 (15%)	62 (34%)	54 (30%)	38 (21%)	182
					861

by the writer or some other member of the full time obstetrics-gynecology staff. The patients were available for follow-up examinations.

We have employed the new International Classification in drawing up these figures, wherein Stage I refers to a tumor confined to the cervix so far as can be told by pelvic examination. Stage II shows some degree of spread of the tumor, but not involving more than the upper third of the vagina and not extending entirely to the pelvic wall. Stage III refers to a tumor involving the parametrium entirely to the pelvic wall or the lower third of the vagina, but not involving the rectum or bladder. If such an involvement occurs, it becomes Stage IV.

It is readily apparent from this table that only one-third of our patients were in the early and, therefore, curable stages previous to 1942; one-half of our patients have been in these more favorable groups since that time. It does seem unfortunate, however, that about one out of every seven patients is still in Stage IV—the hopeless group. That this figure can and should be further reduced is obvious.

We are justifiably proud of our results at the University of Kansas Medical Center when these results are compared with those of other treatment centers in this country (Table II). There will always be some difference of opinion as to the exact stage in which

TABLE II
CARCINOMA OF CERVIX
Results of Treatment

1942-46	*Stage I*	*Stage II*	*Stage III*	*Stage IV*	*All Stages*
Buffalo	159 (70%)	448 (45%)	213 (31%)	154 (16%)	42%
New Orleans	91 (68%)	185 (47%)	389 (25%)	71 (4%)	34%
Sloane & Womens	117 (59%)	137 (30%)	59 (9%)	12 (0)	35%
Jefferson & Penn.	69 (73%)	117 (47%)	87 (23%)	35 (6%)	41%
St. Louis	53 (74%)	92 (42%)	41 (24%)	7 (0)	46%
Stanford & Calif.	121 (67%)	166 (33%)	68 (28%)	18 (0)	42%
Kansas (1942-48)	610 (68%) (84%)	1145 (42%) (58%)	857 (25%) (33%)	297 (9%) (0)	45%

any given patient should be classified when examined by different people. These differences are, however, not nearly so great as formerly. Because of this personal equation, it is argued that over-all rates of cure should be the only consideration, rather than the results in the individual stages of the disease. This is only partially true, since the cure rate in Stage IV is virtually zero, and if one is inflicted with a large proportion of Stage IV patients, he cannot hope to obtain the best over-all rate. The avoidance of late diagnosis and also long lag periods is, therefore, of great importance.

Table III shows the results of therapy over the past

TABLE III
CARCINOMA OF CERVIX
Results of Therapy

	Stage I	*Stage II*	*Stage III*	*Stage IV*	*Totals*
1929-42	32—21 (66%)	113—52 (46%)	190—50 (26%)	100—1 (1%)	435—124 (28%)
1942-48	44—37 (84%)	76—44 (58%)	88—29 (33%)	36—0 (0%)	244—110 (45%)
1949-53	28	62	54	38	182

Too recent to report results.

25 years (except those patients treated in 1949 and later). It is obvious that we have improved our technic and, therefore, our results. A great part of the credit for this improvement should go to Dr. Galen Tice, whose unrelenting efforts at improvement of roentgen therapy have been so important. We are also equally convinced that the accurate placement of radium, with careful individualization of each patient, has paid big dividends. As previously pointed out,[4] careful follow-up with prompt repetitive therapy, when needed, has materially improved our cure rate (4 per cent over-all increase in cures and 10 per cent increase among those requiring additional therapy). This careful follow-up program (only three patients were not followed in the first 500 treated) was started (1) as a labor of love, and (2) to train ourselves in the best possible manner. It has, however, resulted in an increase in cure rate, and it continuously impresses us as being one of the most important items in the treatment of this disease.

The recent report of Dr. Joe Meigs[5] on his experimental surgical approach to early carcinoma of the cervix is worthy of careful consideration. Here we have one of the most expert of all surgeons, selecting only those patients who are good surgical risks, producing results no better than, and sometimes not quite so good as those reported by McKelvey, Norman Miller, and others from radiation therapy, and the series treated by radiation includes patients in all states of physical condition.

It would seem, therefore, (and with this I am sure Dr. Meigs would not disagree) that up to now surgery has less to offer than radiation in the treatment of early carcinoma of the cervix. No one would offer surgery in place of radiation for the later stages. It would seem that the place of surgery, if any, would be in those patients in whom radiation treatment has failed.

The results of the work of Brunschwig, Parsons, and others (pelvic evisceration) are, as yet, not convincing so far as ultimate cure is concerned. The claim that these patients can be temporarily relieved of pain until they die of their disease can well be important. The fact that no expert can always distinguish between the pelvis full of inflammatory induration (so often true of cervical carcinoma both before and after radiation therapy) and the pelvis full of carcinoma, makes this whole problem difficult of adjudication. Many of the evisceration procedures result in removal of inflammatory tissue only—a procedure not so dangerous as it would have been before the antibiotic era. It is obvious that failure of irradiation should not be followed by additional irradiation. It is difficult for the physician to tell such patients that there is no hope and nothing more to be done. Perhaps surgery should be offered to these individuals when death is not imminent. It is equally obvious that such a major surgical procedure as evisceration should not be done on a virtually moribund patient.

The "present status" of treatment of cervical carcinoma would seem to be (1) further education of the public (and physicians) in early diagnosis; (2) further reduction in the lag period between first examination and definitive therapy; (3) application of the most modern methods of pelvic examination, staging of the tumor, accurate radium placement and dosage, and careful, accurately watched, roentgen therapy; (4) meticulous follow-up with possible surgical attack on radiation failures with re-radiation in incomplete results, determined at the end of three to six months and in cases of recurrences discovered as much as 15 years later. We feel that these examinations should be at intervals of several weeks for the first two years, less frequently for the next three years, with one or two examinations per year thereafter. Urinary tract evaluations and chest x-rays should be made on slight indications. We are strongly of the opinion that all these patients should be followed for a minimum period of 15 years.

BIBLIOGRAPHY

1. Heyman, James: Eighth Annual International Report (1952), Radium Hemmet, Stockholm.
2. Payne, Franklin, et al.: Radiation distribution in the treatment of cervical carcinoma, Obst. and Gynec. 1:4, 378-386 (Apr.) 1953.
3. Brown, Willis E.: Effect of radiation on metastatic pelvic lymph node involvement in carcinoma of the cervix, 1951 Trans. Amer. Gynec. Soc. 74:181-197.
4. Calkins, L. A.: Retreatment of carcinoma of the cervix, Southern Med. J. 41:10, 902-906.
5. Meigs, Joe V.: Radical hysterectomy with bilateral pelvic lymph node dissections: Report of 100 patients operated on five or more years ago, 1951 Trans. Amer. Gynec. Soc. 74:164-180.

Spontaneous Intravascular Clotting

William W. Abrams, M.D.
Kansas City, Kansas

Discussions on increased coagulability of intravascular blood are few. In the cases reported, the condition exists usually secondary to some other condition, such as phlebothrombosis following sudden or prolonged rest incident to myocardial infarction, or by pelvic (or lower portion of the body) surgery, or by trauma. Inflammatory conditions as thrombophlebitis and septic emboli have been held to be etiological agents in intravascular clotting.

Recently there have been noted cases of increased coagulability of the blood of patients who have acute pancreatitis. The phenomena which result are different from those in patients in whom there are infarctions, or those in whom circulatory stasis of the extremities tends toward the production of thromboembolism. There are produced changes in the blood itself, brought on by the injection into or absorption of humoral agents from a diseased organ of the body.

Pathologically, in acute pancreatitis there is an edematous pancreas, swollen, and with much hemorrhage and hyperemia throughout. There are usually numerous areas of necrosis of the fat in and immediately adjacent to the gland, and there may be an associated chemical peritonitis, with or without bloody discoloration. Microscopically, there appears to be a metaplasia of the epithelial cells of the pancreatic ducts, the etiology of which seems to be obscure and unproved, plus areas of polymorphonuclear infiltration of the gland. Factors such as chronic alcoholism.[1] regurgitation of infected bile into the pancreatic ducts following obstruction of the latter at the ampulla of Vater (or regurgitation of food into the accessory pancreatic duct), or even a humoral agent, as from acute parotitis, have been implicated in the cause, and cases have been published which have actually shown the apparent connection of the alleged inciting agent and the pancreatitis. It is prob-

able that there is no one of these that has more merit than another, or it is possible that the actual cause has not been found at all.

In its pathologic physiology, the destruction of the gland allows some of the secretions, such as trypsin, etc., to directly enter the blood stream, where it affects the various components already present and elicits an anaphylactic response producing shock.[2] Prothrombin is converted to thrombin, resulting in increased coagulability of the blood intravascularly. It is thought also that trypsin inhibits the action of heparin, thus allowing the clotting of blood to occur more freely. (Paradoxically, there has appeared in the literature recently the treatment of thrombophlebitis with intravenous instillations of trypsin.)[3]

In the cases presented, there are shown the findings of massive intravascular clotting, both venous and arterial, with its profound attendant phenomena and sequellae.

Case No. 1, Providence Hospital No. 53-1004, Mrs. N. A., age 62. The illness of the patient began on January 30, 1953, with the complaint of shortness of breath on exertion and nausea of one day's duration. The patient had arterioslerotic heart disease and arterial hypertension, and her blood pressure that date was 190/102 mm. Hg. Other physical findings that date were auricular fibrillation and extreme obesity. The chest was clear, abdomen was obese, and there was tenderness on palpation in the right upper quadrant. She was seen again at the office on February 3, 1953, because of severe nausea unproductive of vomitus. She apparently improved, and nothing was heard of her until February 11, 1953, when a call was received from a member of the family who stated that the patient had become mentally confused.

On hospitalization that date, she was unable to give a satisfactory history. She complained then of a tenderness behind her right knee. There was a temperature of 101°F.; she was irrational and uncooperative. Blood pressure was 170/100 mm. Hg., pulse was 96, respirations 20, heart rhythm grossly irregular, abdominal obesity, with slight tenderness in the right upper quadrant. There appeared to be a swelling in the popliteal area of the right lower extremity.

Laboratory findings on February 12, 1953, showed the urine to have a specific gravity of 1.020, with a trace of albumin, negative sugar, and 8 to 10 fine and coarse granular casts. The hemoglobin was 13.5 gms., or 87 per cent; the red blood count was 4.48 million. The white blood count was 13,700 with 81 per cent polymorphonuclears, 78 of which were filamented and 3 non-filamented, 16 per cent lymphocytes, 2 per cent monocytes, and 1 per cent basophiles. The sedimentation rate was 16 mm. in 1 hour. On February 21, 1953, the serology was 3 plus. On February 23, 1953, the white blood count was 20,000 with 90 per cent polymorphonuclears. On February 26, 1953, the prothrombin time was control 12.5", patient 26", 19 per cent prothrombin activity.

Roentgenological examination of the chest (portable) on February 12, 1953, showed an elevation of the right diaphragm (slight), no evidence of pulmonary parenchymal infiltration, cardiac size and contour average normal, with the roentgenologist's comment that there was no gross evidence of disease. Electrocardiographic studies this same date showed auricular fibrillation; a repeat electrocardiogram on February 20, 1953, again showed auricular fibrillation, plus bradycardia due to digitalis and the possibility of some disturbance in the anterior coronary artery.

Progress in the Hospital: The mental confusion gradually cleared. She ran a spike-type temperature with daily elevations of up to 101°F., and 102°F. The day after admission the veins behind the right knee became swollen, and cold compresses were applied to the area. She was given 500,000 units of penicillin intramuscularly twice daily, in addition to daily depo-heparin. On February 16, 1953, digitalis was discontinued and quinidine therapy was started in an attempt to control the auricular fibrillation which was thought to be sending off showers of emboli into the circulation. There was noted a beginning cyanosis of the right foot up to the ankle on February 21, 1953. Her blood pressure that date was 126/70 mm. Hg. The next day there was noted extension of the cyanosis to the right forearm and left leg.

On February 24, 1953, the temperature was normal. The patient stated that she felt better, in spite of the condition of her arm and legs. Surgical consultation result was "saddle embolus at bifurcation of the aorta"; medical consultation result was that the patient was having "multpile emboli and arterial occlusion, with possible secondary thrombi, with her cardiac condition preventing surgery (embolectomy)."

The patient expired on February 27, 1953. The pertinent findings at autopsy were: The body was that of an approximately 60-year-old, obese white female 5 feet 10 inches tall weighing approximately 200 pounds. Head, neck and chest showed no abnormalities. The abdomen was moderately distended; no masses could be felt. The lower extremities showed gangrene with swelling and redness extending to an area 6 cm. above the knee.

In section, the heart showed a subendocardial hemorrhage 3 mm. in diameter on the posterior surface of the left ventricle. Atherosclerosis of the coronary arteries, most marked in the descending branch of the left coronary, was present. The lungs presented a thrombus in a blood vessel of the posterior right lower lobe, and the left lower lobe had

numerous vessels filled with antemortem thrombi that completely occluded the vessels. Microscopically, no attachment of the thrombus could be demonstrated. The liver, gall bladder, spleen, kidneys, head, neck, and thyroid showed nothing of note. The pancreas showed areas of yellowish fatty degeneration with focal areas of necrosis. Zones of hemorrhage surrounded some of the necrotic areas. In some instances a purulent appearing material could be expressed from the cut surface. The necrotic lesions were focal but were diffusely spread throughout the pancreatic tissue. No obstruction to the pancreatic ducts could be identified. Microscopically the acinar cells were moderately retracted from their surrounding basement membrane. Most sections contained large areas of fat necrosis with a cellular debris at the periphery in some and an infiltration of lymphocytic cells, along with an occasional polymorphonuclear leukocyte, in others. There was an increase of fibrous tissue surrounding some of the areas of necrosis with proliferation of fibroblasts. In still other sections, the areas of fat necrosis were surrounded by an inflammatory reaction with no fibroblastic proliferation. In several of the sections, hemorrhage was seen in the tissue surrounding the areas of fat necrosis.

The aorta was opened throughout. The wall was of normal thickness. Several small linear lipoidal streaks were identified, but otherwise no unusual changes were seen. The iliac vessels were examined, and a thrombus was present in the left iliac artery beginning at a distance of 5 cm. from the bifurcation of the aorta. The thrombus extended for a distance of 5 cm. into the femoral artery. The lumen in this area was dilated; the walls showed no gross change. The external saphenous, the iliac and the femoral veins, and the popliteal veins contained an antemortem thrombus. The right femoral vein similarly had an antemortem thrombus. At a distance of 1 cm. from the origin of the supraclavicular artery, an antemortem thrombus was present in the brachial artery that completely occluded the vessel.

In one of the sections of the adrenal, a zone of necrosis was noted into which hemorrhage had occurred. In another section the architecture of the cortex was disturbed and replaced by a group of cells which were well circumscribed from the surrounding adrenal parenchyma. These cells had a foamy cytoplasm with basophilic nuclei.

Microscopic sections of the cervix, Fallopian tubes, ovaries, brain, pituitary, and thyroid showed nothing pertinent.

The final diagnosis was: (1) acute fat necrosis of the pancreas; (2) thrombosis of both iliac and femoral veins, the left iliac and femoral arteries, and the right brachial vein artery; (3) gangrene of the distal one-third of both thighs and lower legs; (4) fatal massive pulmonary embolism, left, with areas of infarction of the lower lobe of the left lung; (5) coronary arteriosclerosis; (6) hyperplasia of the right adrenal cortex.

Here was a case of acute pancreatitis that had nausea as the presenting symptom. There was never abdominal pain, and the patient complained of tenderness in the right upper quadrant on deep palpation only. She later complained of swelling behind the right knee, was seen to have gangrene of the

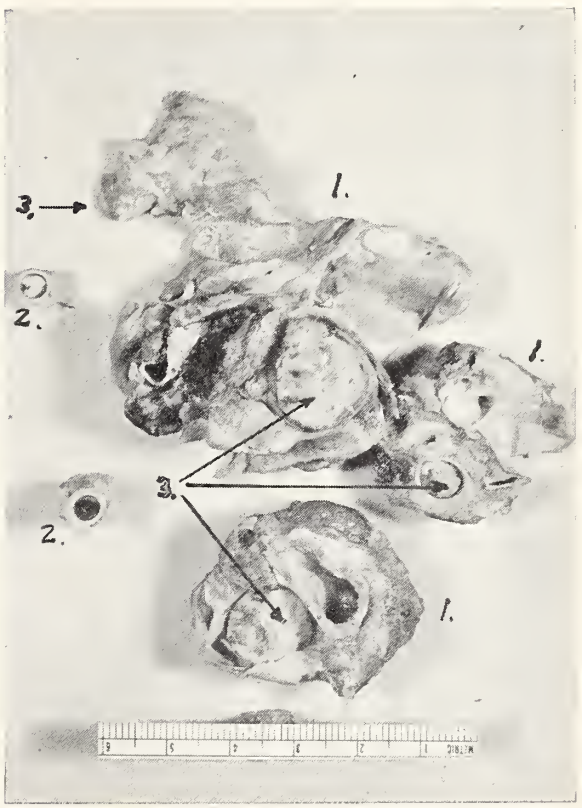


Figure 1. Case 1. Tissue. 1—lung; 2—peripheral arteries with thrombi; 3—thrombi.

extremities, and autopsy showed antemortem thrombi in both arterial and venous vessels. The fact that the coagulation occurred in both arteries and veins shows that a change in the consistency of the blood had occurred so that spontaneous clotting within the vessels took place in different parts of the body simultaneously.

Case No. 2, Providence Hospital No. 54-1382, H. H., age 55. The patient was a male in good nutritional state. On February 17, 1954, the patient called on the phone and stated that he had severe pain in his right upper side; he requested that arrangements be made to have his gall bladder "cut out." He was advised that x-ray pictures and examinations be made to be sure of the necessity of such a procedure, and on February 18, 1954, roentgenological examination

showed a normally functioning gall bladder without stones.

Nothing more was heard of the patient until February 23, 1954, when he again phoned and complained of a constant pain in his right upper abdomen. He stated that he had vomited "a bitter material" a few times. I advised hospitalization, and further history at the hospital revealed that he had had stomach trouble for some years, and that he dated his present condition back 10 days when he noticed his "abdomen beginning to swell."

In the past history there was no gallbladder disease, no ulcers, no alcoholism. Further history, by systems, revaled some shortness of breath on exertion. There was no history of diarrhea, constipation, or genitourinary disease.

Physical examination on admission revealed the patient to be dehydrated, sitting up in bed, with shortness of breath and with severe abdominal distention. He stated that he felt better when he was in sitting-up position. The abdomen revealed a board-like rigidity in the right gastric area and in the right hypochondrium.

The admitting laboratory reports were: urine: color straw, reaction pH 6.0, specific gravity -1023, albumin faint trace, sugar 0, microscopic negative. The red blood count was 5,080,000, with 14.6 gm.—94.2 per cent of hemoglobin. The white blood count was 10,000, with 89 per cent polymorphonuclear cells, filamented 79 per cent, non-filamented 10 per cent, and 11 per cent lymphocytes. Non-protein nitrogen was 32 mg. per cent. The serum bilirubin was .3 mg. per cent "direct" and .3 mg. per cent "indirect." Blood amylase was 42 units. The prothrombin time showed 60 per cent of prothrombin activity. Lipase was 0.3 cc. The sugar tolerance test was:

Fasting 158 mg. Urine neg. 2 hours 235 mg. Urine 4 plus
½ hour 277 mg. Urine neg. 3 hours 175 mg. Urine 4 plus
1 hour 293 mg. Urine 4 plus 4 hours 153 mg. Urine 1 plus

Progress: The patient complained considerably of steady pain in his abdomen; this pain was non-radiating. A Wangensteen tube was inserted because of distention and vomiting. He was given an intravenous solution of 2,000 cc. of 5 per cent glucose in saline and pencillin, one million units plus one gram of streptomycin daily. Demerol, 75 mg., with 1/150 grain of atropine was given for pain. In addition, he was given transfusions.

With the negative laboratory findings to back up a diagnosis of acute pancreatitis (with the exception of the blood sugars), he was taken to surgery for surgical decompression (Dr. M. A. Walker). With the advice of the hospital roentgenologist, who stated that there was a possible obstruction at the splenic flexure, a cecostomy was done for decompression. This was without results, and it was felt then that there may have been a small bowel obstruction instead. The abdomen postoperatively remained distended. The cecostomy was irrigated every six hours. The penicillin-streptomycin injections were continued. The fluid from the Wangensteen came back brownish red, as also did the fluid from the cecostomy. On March 3, 1954, the non-protein nitrogen was 41 mg. per cent. The total proteins on this date were 5.90; albumin 4.05, globulin 1.85.

On March 8, 1954, a repeat roentgenogram of the abdomen showed the small intestines to be filled with gas, suggesting the presence of generalized peritonitis. The roentgenologist stated that the impression was that of ileus, and there appeared to be some fluid in the abdominal cavity. Clinically the patient's pulse ran between 90 and 130, being elevated most of the time, and his temperature was between 102° and 104°.

The condition of the patient deteriorated rapidly; however, he remained conscious and rational. On March 8, 1954, the non-protein nitrogen was 65. Total proteins were 7.40; albumin 3.30, globulin 4.10. The blood sodium which was 144.2 mEq/1 on March 3, 1954, had elevated to 151 mEq/1. Potassium showed no changes as evidenced by the electrocardiogram which also showed a normal sinus rhythm. However, he was given potassium chloride, 20 cc. (2.98 gm.), one ampule, through the nasal tube.

The patient expired on March 9, 1954. The autopsy, with the body being opened with the usual Y-shaped incision, showed the folowing: The peritoneal surfaces were smooth with the exception of the omentum and loops of intestine adherent by fibrinous adhesions to the surgical incision in the right lower quadrant. Adhesions were also present between several loops of bowel and the gallbladder. Heart showed mild atherosclerosis with mild narrowing of the coronary lumina. No thrombi were present. The lungs showed red thrombi in the smaller vascular channels of the left lower lobe. The right lower lobe was firmer in consistency, dark red in color, and on section showed extensive congestion and interstitial hemorrhage. The larger vessels were dilated and contained red thrombi. Histologically, a section through a large pulmonary artery, dilated and containing a red thrombus, was taken. The adjacent pulmonary parenchyma showed extensive edema and hemorrhage in the alveolar spaces with congestion of the alveolar walls. The liver showed nothing of note. The gallbladder showed an inflammatory process, with a lymphocytic infiltration in the mucosa and through the wall. There was also seen necrosis of the overlying epithelial folds with deposition of brownish bile pigment within the necrotic debris. Large foamy phagocytes were present. The extreme

edge of the section showed necrosis of the entire wall with polymorphonuclear leukocytes and a heavy lymphocytic infiltration. The spleen showed nothing abnormal. The pancreas showed areas of moderate increase in fibrous tissue and focal areas of lymphocytic infiltration. In many sections, focal areas of necrosis were present involving pancreatic tissue and the surrounding fat. These areas showed a bluish discoloration with loss of the architecture. In some, an infiltration of polymorphonuclear leukocytes was seen in the adjacent fat tissue with lymphocytes more peripherally. The adrenal, kidneys, bladder, prostate, and gastrointestinal tract showed nothing unusual. The aorta showed thickening of the intima.

The final diagnosis was: (1) acute pancreatic fat necrosis with acute peritonitis, adynamic ileus (first portion of small intestine) and necrosis of the wall of the gall bladder; (2) pulmonary thrombosis with infarction of the lung; (3) recent cecostomy; (4) generalized arteriosclerosis with calcific aortic arteriosclerosis and mild coronary arteriosclerosis; (5) generalized visceral congestion.

The comment by the pathologist was that this patient had a thrombosis in the pulmonary vessels and/or emboli of the same material and subsequent infarction.

Diagnosis of acute pancreatitis is becoming relatively more common. The usual picture of the patient in extreme upper abdominal pain (the patient of Case No. 1 had no pain, but had extreme nausea; it is possible that she felt little pain because of shock), the presence of shock, cyanosis, low blood pressure, rapid pulse and distended abdomen produced by ileus, as was shown in Case No. 2, point to the possibility of acute pancreatitis. Laboratory aids are important, but one must consider the timing and clinical value of the tests. Raffensperger showed that dependence on elevated serum amylase as a diagnostic criterion could be erroneous as various other factors could produce the elevation, namely, peptic ulcer, acute peritonitis, edema of the outlet of the pancreatic ducts, and small bowel obstruction.[4] The amylase test is of real value only in the first 24 hours. Most of the tests shown in the second case were normal with the exception of the glucose tolerance test, which showed an evident damage to the pancreas.

SUMMARY

The additional finding of thrombosis occurring intravascularly in cases of acute pancreatitis is of considerable value, and this should increase our efficiency in diagnosis. The presence of thrombosis in both the venous and the arterial systems simultaneously, early detection of cyanosis or even beginning gangrene of the extremities point to the existence of a change in the character of the blood itself which could have its origin in the pancreas.

BIBLIOGRAPHY

1. Myers, W. K., and Keefer, C. S.: Acute pancreatic necrosis in acute and chronic alcoholism, New Eng. J. Med. 210:1376, 1934.
2. Innerfield, Irving: Acute pancreatitis: the antithrombin titer as an aid in diagnosis and prognosis, New York State J. Med. 52:2239-2243, 1952.
3. Innerfield, Irving; Angrist, Alfred, and Schwarz, Alfred: Parenteral administration of trypsin, J.A.M.A. 152:7, 597-604 (June 13) 1953.
4. Raffensperger, Edward C.: Elevated serum pancreatic enzyme values without intrinsic pancreatic disease, Ann. Int. Med. 35:342 (Aug.) 1951.

The true greatness and the true happiness of a country consist in wisdom, in that enlarged and comprehensive wisdom which includes education, knowledge, religion, virtue, freedom, with every influence which advances and every institution which supports them. —*Henry Giles*

Poisonings with the Newer Pesticide Compounds

Thomas R. Hood, M.D., and Evan Wright

Topeka, Kansas

In the past few years a large number of new pesticides have been introduced and widely used in agriculture, industry, and the home. Many of these pesticides are highly toxic. The problem of identification and treatment is often difficult because of the complicated formulations in which specific toxicants may be found. Kansas law requires the registration of formulas of all pesticides sold in the state. These formulas are on file in the Control Division of the State Board of Agriculture in Topeka. In cases of poisoning where a trade name of a pesticide is known, a call to this department will permit the physician to obtain complete information on the toxicants. If the original container can be located, the label should give a complete formula.

In many cases of poisoning, the nature of the toxic agent or agents is not known. The treatment is, therefore, symptomatic and may include:

1. Emesis and gastric lavage if poison has been taken internally.

2. Evacuation of the gut (avoiding oily laxatives where it is possible that an organic solvent or a halogenated insecticide is involved).

3. Thorough washing of the eyes or body if there has been external contact with the poison—wear rubber gloves while aiding the patient.

4. Supportive therapy.

A. Sedatives. Sodium pentobarbital is preferred for acute poisoning because of its rapidity of action. Phenobarbital is useful in maintaining a prolonged level of sedation in persisting hyperexcitability or convulsions.

B. Stimulants. In treating vascular collapse, substances such as adrenalin should be used only after careful consideration, as they are contraindicated in poisoning by the halogenated hydrocarbons.

C. Transfusions. In patients *in extremis* or extended syncope, transfusions may be indicated except where pulmonary edema is already present. If blood is not readily available or if dehydration is suspected, 5 per cent glucose or normal saline infusions are indicated.

D. Oxygen therapy. Conventional oxygen-carbon dioxide mixtures should be administered to patients showing cyanosis or severe respiratory difficulty. Patients with pulmonary edema require oxygen under positive pressure as well as postural drainage and dehydration therapy until the exudate is checked.

Dr. Hood is executive secretary of the Kansas State Board of Health. Mr. Wright, also with the Board, is director of the Food and Drug Division.

INSECTICIDE FORMULATIONS

Insecticides for agricultural and industrial use may be prepared in many ways. They may be the pure toxicant substance in liquid, crystalline, or powder form. They may be solutions in water or hydrocarbon solvents, emulsions, dusts, wettable powders, or suspensions. Household insecticides may be powders, dusts, solutions in either water or hydrocarbons. The popular "bug bombs" are solutions of the toxicants in hydrocarbons, usually deodorized kerosene, with a Freon propellant to produce an aerosol. Synergists or activators are often added to the formulations to give greater killing power.

For greater convenience, the most common of the newer insecticides may be divided into two types—the chlorinated hydrocarbons and the organic phosphates.

THE CHLORINATED HYDROCARBONS

DDT	Dichloro-diphenyl-trichloroethane
TDE	Dichloro-diphenyl-dichloroethane
DFDT	Difluoro-diphenyl-trichloroethane
Methoxychlor	Dimethoxy-diphenyl-trichloroethane
BHC	Technical benzene hexachloride
Lindane	Gamma isomer of benzene hexachloride
Toxaphene	Chlorinated camphene
Chlordane	1,2,4,5,6,7,8,8-Octachloro-2,3,3a,4,7,7a-hexahydro-4,7-methanoindene
Aldrin	1,2,3,4,10,10-Hexachloro-1,4,4a,5,8,8a-hexahydro-1,4,5,8-dimethanonaphthalene
Dieldrin	1,2,3,4,10,10-Hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1,4,5,8-dimethanonaphthalene
Heptachlor	1,4,5,6,7,8,8-Heptachloro-3a,4,7,7a-tetrahydro-4,7-methanoindene
Prolan	2-Nitro-1,1-bis(p-chlorophenyl) propane
Bulan	2-Nitro-1,1-bis(p-chlorophenyl) butane

Routes of Absorption: All the chlorinated hydrocarbons are absorbed through the skin as well as from the gastrointestinal system and respiratory system. The compounds which are normally liquids are in general absorbed through the skin more rapidly than those which are normally solids. Solutions in oily or hydrocarbon solvents are more actively absorbed than dry formulations.

Symptoms: In general the chlorinated hydrocarbons are central nervous system stimulants. The mechanism of their action is not known. Symptoms most frequently experienced are hyper-excitability, irritability, loss of equilibrium, clonic-tonic convulsions. Depression sometimes follows other symptoms. Loss of appetite is a common symptom.

Treatment: Treatment is essentially the same for poisoning by any of the chlorinated hydrocarbons.

Wash the contaminated skin thoroughly with soap and water. Avoid cleaning skin areas with such materials as benzene, gasoline, or carbon tetrachloride. Clear the alimentary tract if the poison has been taken internally, using gastric lavage and saline laxatives and avoiding oily laxatives. Sedation to restore a relative calm is necessary, the drugs of choice being sodium phenobarbital, phenobarbital, and pentobarbital. Paraldehyde and urethane have been used effectively. The object of sedation is not to induce sleep but to counteract excitability, and it should not be carried to the extent of interfering with eating. However, large doses of the drugs may be given and pushed to the limit of therapeutic effectiveness. Persons poisoned by stimulants may tolerate large doses of barbiturates without undue depression and with great benefit. Calcium gluconate, intravenously, has been reported to have some effectiveness as an antidote, and, as its mode of action is different, it may be used in conjunction with barbiturate therapy.

THE ORGANIC PHOSPHATES

Parathion	O,O-Diethyl-p-nitrophenyl-thiophosphate
Dimethyl parathion	O,O-Dimethyl-p-nitrophenyl-thiophosphate
Paraoxone	O,O-Diethyl-p-nitrophenyl-phosphate
OMPA	Octamethyl pyrophosphoramide
TEPP	Tetraethylpyrophosphate
HETP	Hexaethyltetraphosphate
ASP-47. Sulfatep	Tetraethyldithionopyrophosphate
Malathion	O,O-Dimethyl dithiophosphate

The uses of the organic phosphates are almost entirely agricultural, although a small amount of parathion has been sold for domestic garden use.

Routes of Absorption: The organic phosphates are readily absorbed through the skin. They may be absorbed through the respiratory system from the inhalation of sprays. Ingestion would be equally dangerous. Most of the poisonings which have occurred have been through spillage of the concentrated chemicals on the skin, or through combined skin exposure and inhalation of sprays. The organic phosphates act as irreversible inhibitors of the enzyme, cholinesterase, and thus allow the accumulation of large amounts of acetylcholine. When the critical level of cholinesterase depletion is reached, symptoms appear. Recovery is apparently complete if there is time for the reformation of the critical quota of cholinesterase; however, additional exposure before recovery is complete has an additive effect.

Symptoms: Symptoms of organic phosphate poisoning include headache, giddiness, blurred vision, weakness, nausea, cramps, diarrhea, and discomfort in the chest. Signs include sweating, miosis, salivation, pulmonary edema, cyanosis, papilledema, convulsions, coma, and loss of reflexes and sphincter control. The last several signs are seen only in advanced cases but do not preclude a favorable outcome if energetic treatment is continued. The onset of symptoms may be quite rapid, and treatment should begin as soon as exposure is recognized.

Treatment: Keep the patient completely atropinized if symptoms appear. Give 1 to 2 mg. (1/60 to 1/30 grain) per hour up to 10 or 20 mg. in a day, particularly for the control of respiratory symptoms. The intravenous route is most rapid. It will be noted that this dosage of atropine is greater than that ordinarily employed for other purposes. Morphine, theophylline, and aminophylline are contraindicated.

If the patient has not yet shown symptoms or if they have been allayed by the first dose of atropine, he should be completely and quickly decontaminated. Wearing rubber gloves, remove the patient's clothing, and with due regard for his condition at the moment, bathe him thoroughly with soap and water. If washing soda is available, use it, for the organic phosphates are hydrolyzed more rapidly in the presence of alkali.

If there is any suspicion that an organic phosphate has been ingested, induce vomiting, give some neutral material such as water, and induce vomiting again.

If pulmonary secretions have accumulated before atropine has become effective, the patient should be turned to a suitable position for postural drainage of mucus. Suction should be used as needed to remove secretions. If the stomach is distended, it may be emptied with a Levine tube.

The use of an oxygen tent or even of oxygen under slight positive pressure is advisable and should be started early. The patient should be watched constantly for the appearance of muscular weakness, and the need of artificial respiration may appear abruptly. Equipment for artificial respiration should be placed at the patient's bed in readiness as soon as he is hospitalized. Complete recovery may occur even after many hours of artificial respiration have been necessary.

The acute emergency lasts 24 to 48 hours, and the patient must be watched constantly during that time. Favorable response to one or more doses of atropine does not guarantee against sudden and fatal relapse. Medication must be continued through the entire emergency.

Following exposure heavy enough to produce symptoms, further organic phosphate insecticide exposure should be avoided. The patient remains susceptible to relatively small exposures of the phosphates until regeneration of the cholinesterase is complete.

THE HYDROCARBON SOLVENTS

Kerosene, deobase, coal oil—Kerosene is a mixture of aliphatic hydrocarbons distilled from petroleum in the temperature range of 204 to 315 degrees F.

Toluene, toluol, methylbenzene.
Xylene, Xylol, dimethylbenzene—A mixture of *o-*, *m-* and *p-* xylene.

Kerosene, particularly the deodorized type (deobase) is used extensively as the solvent and vehicle for insecticides in household flysprays. It is also used as a solvent in agricultural sprays and bases for emulsions. Xylene is a common solvent in insecticidal solutions, emulsion concentrates, and emulsions. Toluene is used to a lesser extent than xylene for the same general purposes.

Routes of Absorption and Symptoms: In any poisoning resulting from a formulation of insecticide containing any of the hydrocarbon solvents, greater danger to the patient may lie in the solvent rather than in the solute toxicants. This is particularly true if any of the material has been aspirated. In the case of kerosene, it has been shown that absorption from the gastrointestinal tract is not significant as a cause of mortality, but that the small quantity necessary to produce fatal poisoning by aspiration should be emphasized. Ingestion frequently results in immediate gagging and coughing with consequent aspiration of the oil. The initial symptoms are followed by deep drowsiness. In more serious cases, bronchopneumonia may develop in 24 to 36 hours. Xylene and toluene are apparently absorbed from the intestinal tract, and because these substances in technical grades usually contain some benzene, it must be considered that either ingestion or inhalation may affect the hemopoietic system as benzene.

The use of sprays made with hydrocarbon solvents in closed spaces may lead to headache, disturbances of vision, dizziness, poor coordination, and nausea. More severe exposure may result in collapse and coma before the patient is aware of his condition.

All the hydrocarbon solvents are irritating to tender skin and other tissues. Absorption through the skin is not significant under ordinary conditions of exposure. Xylene is particularly irritating to tender skin and to the eyes.

Treatment: If eyes and skin are contaminated, they should be thoroughly washed. Two per cent butyn ointment may be placed in the eyes, and any analgesic ointment may be used on irritated skin areas. If solvent has been ingested, an effort should be made to remove it by gastric lavage. In case of kerosene this is a precaution against regurgitation and aspiration, rather than to prevent absorption from the gastrointestinal tract. Saline laxatives may be used, but oily laxatives should be avoided, especially if the ingested material contained any insecticides. Sedatives or stimulants may be used symptomatically in moderation. Chemotherapy does not benefit kerosene pneumonia but may be useful in preventing bacterial invasion. Liver damage may be minimized by the use of a low-fat, high-protein diet.

Before leaving the field of insecticides, it should be pointed out that many of the old insecticides, particularly compounds of mercury, lead, arsenic, and fluorine, are still used rather widely. Further, there are a number of new insecticides and adjuvants which have toxicity somewhat lower than those mentioned above, but which, under unusual circumstances, cause poisoning. These include the organic thiocyanates.

RODENTICIDES

Several effective rodenticides have been developed in the past few years. They are sold under trade or coined names: 1080, warfarin, pival, castrix, and Antu. These products have almost entirely replaced the phosphorus, arsenic and strychnine, and squill compounds formerly used to poison rodents.

1080, Ten Eighty—Sodium Monofluoracetate. Ten eighty is extremely toxic to all forms of animal life. Because of its great toxicity it is not available for domestic use. It is available in Kansas only to licensed pest control operators placed under bond.

Routes of Absorption and Symptoms: Ten eighty is not readily absorbed through the skin, but dusts of the crystalline material are readily absorbed through the respiratory system. Absorption is complete through the gastrointestinal tract on ingestion of solutions.

Ten eighty acts through direct interference with the acetate metabolism and has a strong effect on the cardiovascular and nervous systems. Symptoms appear in from 30 minutes to 2 hours or more after ingestion. The first indications of poisoning are nausea and mental apprehension followed by epileptiform convulsions. After a period of several hours, pulsus alternans may appear followed by ventricular fibrillation and death. Children appear to be more subject to cardiac arrest than to ventricular fibrillation.

Treatment: Immediate emesis and gastric lavage followed by oral doses of magnesium sulfate are useful. Administration of certain substances capable of supplying acetate ions has shown antidotal effects in animals, including monkeys; the choice drugs are monoacetin (glycerol monoacetate) (2-4 gm./kg.) and a combination of sodium acetate and ethanol (2 gm./kg. of each). A single dose of magnesium sulfate (800 mg./kg.) given intramuscularly as a 50 per cent solution has saved the life of rats dosed with lethal amounts of 1080. Complete quiet and rest are indicated, but barbiturate anesthesia has proved disappointing when used as an antidote against this poison.

Warfarin, Compound 42, WARF-42 — 3-(α-phenyl-β-acetylethyl)-4-hydroxycoumarin
Pival — 2-pivalyl-1, 3-indandione

Routes of Absorption: Warfarin and pival are readily absorbed from the intestine, and apparently no other route is important. Both rodenticides are

essentially anticoagulants. They inhibit the formulation of prothrombin and apparently produce some capillary damage so that the slightest trauma results in hemorrhage. A single dose probably could not be fatal to man, but several small doses over several days would produce death. Poisoning could not be expected from these rodenticides except where suicide or homicide is intended. The action of these rodenticides is similar to that of Dicumarol, except that capillary damage (the so-called "toxic factor") is greatly increased.

Treatment: After blood has been taken for prothrombin and other tests, a blood transfusion should be given at once if there is reasonable assurance that warfarin or pival poisoning has occurred, irrespective of whether or not signs and symptoms are present. Vitamin K in a dose of 65 mgs. (gr.i) repeated three times on the first day of treatment is suggested, irrespective of symptoms, and smaller doses should continue until prothrombin time has reached normal. In a more seriously ill patient, small transfusions of carefully matched whole blood should be given daily until the patient returns to normal.

The progress of the patient should be followed by the prothrombin test. Tests should be made at least twice daily until a return to normal is clearly established.

Antu.—Alpha-naphthylthiourea.

Antu has now been replaced to a large degree by the safer warfarin and pival. It is still a constituent of some rodenticides used for household rodent control.

Routes of Absorption and Symptoms: Antu has no significant toxicity except when ingested. It kills by the production of pleural effusions, by an action not presently known. It has an emetic action, and normally most of the material will be vomited before serious poisoning occurs. It is especially toxic to rats because they cannot vomit.

Treatment: No antidote is known for Antu. Treatment must be entirely symptomatic.

SUMMARY

This article was assembled for publication with the thought that data relating to clinical aspects of toxic pesticides, rodenticides, and solvents might be a useful reference in the hands of practicing physicians.

Acknowledgment: The material for this paper was derived from a number of sources, many of them manufacturers' information and instruction sheets. Some of this information is so general in distribution it cannot be identified as to its original sources. Special acknowledgment should, however, be made of the use of material from Clinical Memoranda on Economic Poisons, Communicable Disease Center, United States Public Health Service, Savannah, Georgia, and the following series of papers published in the Quarterly Bulletin of The Association of Food and Drug Officials of the United States:

Toxicology of the newer agricultural chemicals, by Arnold J. Lehman, M.D., XII (July) 1948.

Pharmacological considerations of insecticides, by Arnold J. Lehman, M.D., XIII (April) 1949.

Chemicals in foods: a report to the Association of Food and Drug Officials on current developments, by Arnold J. Lehman. M.D.: Part II, Pesticides, XV (October) 1951; Part II, Pesticides. Sec. II, Dermal Toxicity. XVI (January) 1952; Part II, Pesticides. Sec. III, Subacute & Chronic Toxicity, XVI (April) 1952; Part II, Pesticides. Sec. IV, Biochemistry, XVI (July) 1952; Part II, Pesticides. Sec. V, Pathology, XVI (October) 1952.

Pesticides: some chemical considerations and toxicological interpretations, by W. I. Patterson and A. J. Lehman, M.D., XVII (January) 1953.

Distribution and adsorption characteristics of vaporized lindane, by W. A. Queen, XVII (October) 1953.

Toxicological evaluation of household insecticides, by Arnold J. Lehman, XVIII (January) 1954.

The educational process is a universal and lifelong thing. From the first consciousness of the child to the clouded darkening of the dying man's mind it goes on. It would go on, in one way or another, if there were no schools, but there are many urgent factors that call for the best effort possible to teach our young. There are the indescribable riches of the world without and within, the inexhaustible store of past human thought and experience, the turbulent present with its significant experiences, the swift passage of a human life and the long tomorrow of eternity.

—*John E. Riley*

The Doctor Goes to Court

Joseph Cohen, LL.B.

Kansas City, Kansas

So you have been asked to be an expert medical witness. If you are experienced in medical legal testimony, then what I have to say tonight may be old to you. But if you only occasionally grace our courtrooms as expert witnesses, I may then have in the lecture to follow a great deal of information which may prove valuable. On the other hand, even if you are an experienced witness, you may find a number of things in this paper which will help to explain the examination and cross-examination, the objections thereto, and the drama that is part of every trial.

I firmly believe that one of the reasons so many physicians and surgeons shy away from the courtroom, aside from the question of economics involved, is that they do not fully understand and appreciate the role of the medical expert in litigation. After they have apparently given a forthright chronological report of the history, the chief complaints, the clinical examination, the x-ray examination, the treatment given to the patient, the diagnosis, and prognosis, some physicians consider it not only annoying and aggravating but a show of unnecessary hostility when the attorney on the other side of the case undertakes to explore the truth and the reasonableness of that testimony by means of a vigorous, searching, and minute cross-examination.

If we understand at the outset that the principal purpose of litigation is to arrive at the truth, and that counsel on each side of the case is trying as skillfully as he knows how to present the facts to the judge or jury in the light most favorable to his client, then you will have no difficulty in understanding the judicial courtroom process.

If you find the lawyers difficult at times, remember that they, too, are sometimes baffled at the difference between the hospital room statements of the medical profession and the courtroom statements which they hear from the witness stand. It is sometimes hard to understand why a patient will be prescribed a course of treatment for an ailment by the doctor who will be heard to testify for the defense that there is nothing wrong with the claimant because that doctor can find no objective evidence of disability. Why, wonders the attorney, will he prescribe massage and physiotherapy for an ailing back and then take the witness stand later on and testify that there was nothing wrong with the patient?

As we enter the atomic age with the complexities of our modern civilization, our highly mechanized industries, speeding motor cars, and jet propelled planes, we encounter a greatly increased number of cases involving personal injuries. Thus, the role of the physician as an expert witness becomes more and more important.

I believe it makes sense when I say that a physician has no right to decline to give his services to a patient involved in litigation and at the same time expect to be favored with that patient's medical business and that of his family and friends when they are not involved in litigation. Leaving out of consideration the fact that members of the medical profession are not immune from compulsory process to bring them into court, a method seldom used for obvious reasons, there is a strong moral obligation on the physician to deal fairly with his patients and with the courts.

Once we break down the confidence of the people in the judicial process, without offering them a better substitute, we tear down the structure of democracy which has given us the highest standard of living and more civil rights and liberties than are enjoyed by any other people on the face of the globe.

The medical profession has a monopoly which cannot be invaded. If we are to keep a free, voluntary society, if we are to stop tendencies toward socialization, if we are to avoid concentrating the medical legal work in the hands of a few, and not always the most competent in the field, it becomes highly important that each man or woman in the medical profession be willing to come to court and testify.

I can say to you men of the medical profession that you are going to find in the future a much better prepared bar in the medical legal field than we have had in the past. Negligence and workmen's compensation cases comprise by far the largest amount of litigation in the courts. Lawyers are learning that they must have specialized skill and training in this field if they are to do a good job on behalf of their clients. Many of us have for years delved into the medical books and journals in the preparation of our cases in order that we might meet on more equal ground the doctors whose opinions we must test in the crucible of litigation.

Now a number of the leading universities are hold-

Although the JOURNAL ordinarily publishes in this section only articles of scientific content, this address seemed to contain sufficient information of value to a large group of physicians to make its publication here advisable. Mr. Cohen presented the paper before the Wyandotte County Medical Society on January 19, 1954.

ing seminars on the graduate level for practicing attorneys, and in connection with the seminars many leading men of the medical profession are giving of their time and talent to the education of the bar. My son who is a second-year student at Harvard Law School tells me that this year the third-year law students go to the Harvard Medical School where members of the faculty lecture to them on medical legal testimony. One of the leading universities in this field is Texas University, and another leader is Tulane. Our own law school at the University of Kansas City also is holding annually a three- to five-day lecture series given by recognized men in the medical profession.

Therefore, I say to you that the younger lawyers are coming out from the law schools better prepared than before, and we older practitioners are trying not to lag behind. I would like to see the University of Kansas School of Medicine make a distinct contribution in this field. If it would sponsor a course for law students and practicing attorneys, there would be reciprocal advantages to the bar and the medical profession and, of course, to the public.

Your function in the judicial process, as medical experts, is to give your best opinions, based upon your education, knowledge, and experience, on the subject matter before the court. Anybody can testify as to what you prescribed and did for the patient, but only you can testify as to why you did it.

Before you are permitted to testify as an expert, counsel offering you as his witness must qualify you. To do so, he inquires as to your educational background, when you received your degree or degrees, when you started to practice, whether you are a specialist or a general practitioner, what professional organizations you belong to, and sometimes whether you have held official positions in those organizations, your postgraduate training, and the states where you are licensed to practice. The purpose of eliciting your qualifications is not then to determine your skill or want of skill nor to parade before the court or jury the vastness of your medical learning, but merely to lay the groundwork or foundation which will permit you to testify as an expert (*A. T. & S. F. R. Co. vs. Sage,* 49 Kan. 524).

Many times you will hear opposing counsel say that he waives the qualification of the witness. Such a waiver may dispense with the necessity of having you testify as to your educational background. On the other hand, opposing counsel may prefer to qualify his expert witness in order to show the court or jury that he has brought into the courtroom a person of unusual training, and the judge will permit him to do so even though the qualification has been waived. If you are a specialist in your field, the lawyer will want to show the extent of your specialized training. Having qualified, you are then permitted to testify what you have seen, heard, or done for the patient, the history, complaints, diagnosis, treatment, and prognosis. You may be asked for your opinion as to the permanent nature of the disability or whether it is of a temporary nature or has been entirely cured. If there is permanent disability, you may be asked in a workmen's compensation case to give your opinion as to the percentage of disability which the injured workman has suffered.

Such a question always deals in the inexact field of opinion. However, for the purpose of the law, you are asked to assume and take into consideration the amount of work which an ordinary, normal, well workman might perform in the general field of labor, and then determine the percentage of loss the injured workman has suffered from his ability to do and perform general manual labor. No one realizes more than I do the great responsibility which such a question poses to every man in the medical profession who is asked to make such a rating.

Our courts have said that a medical expert need not answer questions with certainty. All he need do is give his opinion (*Roark vs. Greene,* 61 Kan. 299).

Your opinion on cross-examination may be tested by opposing counsel in a number of different ways, depending upon his resourcefulness, ingenuity, and skill. You may be asked whether you agree or disagree with certain statements appearing in recognized medical books and authorities, and for that purpose counsel may read to you excerpts from such books. These medical books are not admissible in evidence to establish the declarations or opinions which they contain (*State vs. Baldwin,* 36 Kan. 1). However, the Supreme Court said (*Stout vs. Bowers,* 97 Kan. 33, 36):

"One of the recognized methods of testing the knowledge of an expert witness who founds his opinions on standard medical authorities is to read from those authorities upon the subject in question and interrogate him as to whether his opinions coincide with those expressed in the books and whether there is not a conflict between the opinions he then gives and the views expressed by the authorities upon which he relies for information."

Before you have completed your direct or cross-examination on the witness stand, you may be asked a hypothetical question. Such a question must include only facts which have been established in the evidence directly or by inferences fairly deducible from established facts. Counsel may not assume the existence of matters material to the formation of a correct opinion about which no testimony has been given previously (*Davis vs. Travelers Insurance Company,* 59 Kan. 74; *Western Union vs. Morris,* 67 Kan. 410). However, counsel may base his hypothetical question on weak and inconclusive testimony of one or more witnesses or inferences properly

deducible therefrom (*Roark vs. Greene, supra.*).

It is not necessary that the hypothetical question incorporate all facts supported by the evidence, nor need it include all facts or all theories advanced by the adversary (*New York Life vs. Daerksen,* 75 Fed. 2d 96). Counsel for plaintiff may frame his hypothetical question on facts and theories tending to support his thesis, and he cannot successfully complain if defense counsel in his hypothetical question incorporates only the facts and theories which he desires to support (*Wingfield vs. McClintock,* 85 Kan. 452).

When you are confronted with a hypothetical question, it is well that the physician bear in mind that he is asked to assume the truth of the facts stated therein, and to give his opinion based thereon without interpolating or questioning these facts. It is unimportant so far as that hypothetical question is concerned whether you disagree with the facts or the theory upon which it is predicated. You have a right to rely on the skill of opposing counsel to bring out and stress both sides of the case. Many physicians inexperienced in courtroom tactics find such hypothetical questions difficult and want to argue with counsel about the facts stated therein. Often they have had to be admonished by the court not to do so but to answer the question as propounded.

It has been my experience that the most effective medical expert testimony is given by the physician who is frank and honest and who will concede points to the opposition when they are entitled to such a concession. A recent survey made by one of the legal publications shows that the least effective expert is the one who finds it impossible to concede any point to the other side for fear that he will weaken his own opinion before the court or jury. Our court has said that an expert witness is to be given such credence and weight as the court or jury believes he is entitled to (*Santa Fe vs. Thul,* 32 Kan. 255; *Baird vs. Shaffer,* 101 Kan. 585).

Objections by opposing counsel to a hypothetical question must be specific and must point out to the court specifically in what way the question is deficient. Usually he will point out that it incorporates certain facts not proven, or misstates proven facts, or makes inferences not justified from the established proof. Only such specific objections may be considered by the court. Young, inexperienced lawyers often must learn this rule the hard way after their general objections are overruled by the court (*Linscott vs. Hughbanks,* 140 Kan. 353, 357).

A medical expert need not feel under tension when subjected to cross-examination. Nor should he take offense if counsel tries to weaken the effectiveness of his testimony. Perhaps the soundest and safest attitude is that which men of science should always adopt—that is that you are aiding the triers of the facts to arrive at the truth. While you would be less than human if you did not want to see your opinion prevail in a courtroom, and perhaps you would be lacking in a good sense of justice if you did not seek to persuade a jury or court to adopt your opinion, on the other hand, having given your opinion, you have performed your full duty and you must then leave the advocacy to the lawyers.

X-rays, cardiograms, encephalograms, and other mechanical aids to diagnosis and treatment are common to your profession, but are not so well known by the bar and certainly not well understood by the layman. X-rays and cardiograms have to be taken and interpreted by men with special skill in that field. Therefore, unless your qualifications indicate that you possess such skill, you may not be permitted to testify as to what the x-rays or cardiograms reveal. On the other hand, if you intend to testify concerning x-ray or cardiographic findings, the x-rays and cardiograms must be brought into court, properly identified, and introduced in evidence so that you may be subjected to cross-examination thereon, and in order that opposing counsel might have the films examined by other men in the profession to test your correctness thereon (*Lefebvre vs. Western Coal Co.,* 289 p. 456).

I might say here that these diagnostic aids are so common to your profession now that if you fail to use them in cases treated by you where indicated, you may be subjected to a claim of malpractice, and it will be no defense for you to claim that the patient did not request that you use an x-ray or take a cardiogram. That was the finding of our court (*James vs. Grigsby,* 114 Kan. 627), where the physician was charged with malpractice in the setting of a broken leg without the use of an x-ray.

Hospital records are always important in a law suit. These are admissible in evidence only when the physician, internes, and nurses identify the records as records made by them in the course of the hospitalization of the patient (*Bowman vs. Coyle,* 124 Kan. 492, 495). In order to avoid such a circuitous manner of establishing the veracity of hospital records, counsel many times will agree between themselves that the record may be introduced in evidence without such qualifying proof.

Those who operate hospitals for the benefit of the public must realize that they, too, are part of the judicial system in this democracy, and even though there is some hardship imposed upon hospitals and some expense, too, it is not asking too much to request that they bring, through their librarians and record clerks, these records into court for examination and testimony. As for myself, I try to see to it that the record clerk is reimbursed to the extent of her taxicab fare when she comes to court with the

record, and I am sure that every thoughtful lawyer would consider that a minimum requirement of co-operation on his part.

Your own private records in these cases are also admissible in evidence, and you must be prepared to bring them into court with you for the purpose of refreshing your own recollection about the case, and you must also be prepared to allow opposing counsel to examine records in the hope that he might find therein something contradictory to what you have previously testified.

The fact that you are a member of a busy profession, that of administering to the weaknesses and ills of humanity, does not give you any immunity from compulsory process. But an attorney should and must respect the demands made on your time, should give you notice of the time when you will be needed, and will excuse you from attending at the specified hour if an emergency makes your attendance impossible. On the other hand, your co-operation is needed and should be whole-heartedly given in order that the judicial branch of government might function properly.

There is adequate power in the law to compel you to attend after you have been served with a subpoena, but I assure you that no lawyer would deem it a pleasant task to send the sheriff after an expert witness who adamantly refuses to obey the process served on him. I learned as a young lawyer by bitter experience than an unwilling, unco-operative medical witness can do more harm than good if he is compelled to come to court against his will. Having subpoenaed a doctor to testify in a deposition at Wichita, and being confident that he could testify only one way because he had already given a statement and signed a death certificate as to the cause of the man's death, I insisted that the physician give his testimony.

I probably would have not done so had this physician not insisted on the payment of an unreasonable fee in advance of his testimony. You can imagine how surprised I was when the physician made a complete about face in his testimony, stating that after full consideration of all the facts in the case, he had decided that the death was not due to food poisoning but was due to some other cause. Probably I would have been smarter had I paid the unreasonable fee demanded by this unscrupulous practitioner.

Privileged Communications. Section 60-2805, General Statutes of Kansas, 1949, sets out the various professional groups who are not privileged to testify concerning communications arising in the course of their professional conduct, such as attorneys and clients, priest and parishioner, physician and patient, etc. It states that a physician or surgeon is incompetent to testify concerning any communication made to him by his patient with reference to any physical or supposed physical disease, defects, or injuries, or the time, manner, or circumstances under which the ailment occurred or concerning any knowledge obtained by a personal examination of any patient without the consent of the patient. However, it further provides that if a person, without objection on his part, testifies concerning any such communication, the physician communicated with may also be required to testify on the same subject as though the consent had been given.

That means that you as a physician or surgeon cannot testify as to these communications which come to you in the course of your professional undertaking, but if the patient files a claim or brings a law suit and testifies concerning his condition, then you can be compelled also to testify thereto because the privilege is then waived. After a patient's death, the heirs or personal representatives may waive the objection (*State vs. Pullman,* 85 Kan. 237, 238; *Gorman vs. Hickey,* 145 Kan. 54, 61, 63). On the other hand, if no heir or personal representative of the deceased objects to your testimony, you may testify for the reason that this law is not a privilege given to the physician, but it is a privilege given to the patient (*Doty vs. Ice Company.* 118 Kan. 323; *Kirsch vs. Federal Life Insurance Company,* 149 Kan. 309, 313).

Malpractice. The obligation of a physician and surgeon is merely to use such skill and foresight in the practice of his profession as is generally used by men in the profession. A physician does not impliedly contract to cure his patients (*Tefft vs. Wilcox,* 6 Kan. 46). In case of doubt on which of two courses to follow, all the physician is required to do is to use his best judgment, and he does not guarantee that he will get good results; if he has bad results he is not subject to civil liability (*James vs. Grigsby,* 114 Kan. 627; *Paulich vs. Nipple,* 104 Kan. 801). In one case (*Rainey vs. Smith,* 109 Kan. 692), our court said that an action for malpractice would lie where a wrong diagnosis was made of the plaintiff while she was pregnant. On the other hand, an honest mistake in diagnosis made where you do not treat the patient does not render you liable. In another case (*Bugg vs. Security Benefit Association,* 173 Kan. 522), a physician was sued for damages for having erroneously diagnosed cancer of a breast. He did not perform the operation for the removal of the breast, and our court said he could not be held liable for this woman's damages because the woman had voluntarily had the breast removed by someone else which was the proximate cause of her injury.

However, in another case (*Russell vs. Newman,* 116 Kan. 268), the Supreme Court ordered a new trial where the jury granted the patient only nominal damages for injuries arising when a sponge was left in an incision after removal of a kidney, in spite of the fact that there was testimony that no injury was

suffered by plaintiff as a result thereof. The court said that the patient was entitled to more than just nominal damages for such a mistake.

On the other hand, I want to advise you physicians and surgeons that you cannot collect your fee for services rendered where the claim of malpractice is successfully raised (*Abbott vs. Mayfield,* 8 Kan. App. 387). However, my experience proves that you do not have too much to worry about from this angle in Kansas for the reason that want of skill in treatment, diagnosis, and operation on a patient is a matter which only men of your profession may testify as to in court (*Waddell vs. Wood,* 158 Kan. 469). Only in rare and unusual circumstances is it possible to get any physician to take the witness stand against a brother member of his profession where that question is at issue. It is not the purpose of this paper to delve into the ethics or propriety concerning the screen of protection which the men of your profession throw around each other when your skill is being tested by the judicial process.

If a case can be proven against you, you are liable in civil damages not only for your own mistakes but for the mistakes of the technicians, assistants, and nurses in your employ, but you are not liable for any carelessness or negligence by the nurses, attendants, or assistants in the hospital where your patients are sent. Non-profit hospitals, such as those operated in Kansas City, Kansas, are no longer immune to civil liability for damages arising out of the negligence of their employees (*Nicholson vs. Hospital Association,* 97 Kan. 480), since the decision in *Noel vs. Menninger Foundation,* 175 Kan. 751. Employees themselves may be subjected to claims, but this is seldom done because in most cases the employees are not financially responsible and do not carry insurance against such liability.

After almost 29 years of a busy trial practice, much of it in the field of negligence and workmen's compensation, when I come in contact with and listen to the testimony of many doctors, I have learned to classify them not as honest or dishonest, stupid or brilliant, forthright or inconclusive, but rather as pessimists or optimists. When I give to the men in your profession proper understanding and sympathy, I realize that the classification of pessimists and optimists is about as good a classification as one can find.

The pessimist is the best kind of expert medical witness from the plaintiff's point of view. He will take a rather dreary look at the patient's chances for recovery, find good reason for the complaints which the patient makes of pain and suffering, and agree that the percentage of disability which the plaintiff will suffer will be great. The plaintiff's bar loves these pessimists.

The optimist works for the insurance companies and big corporations. The patient is never as sick as he says he is. His complaints of pain and disability are not sustained by the findings. He believes that if the patient would just forget about his pain and go back to work, he would recover sooner. As an optimist, he sees no reason why the injured patient should not be doing a full day's work or, in any event, he minimizes the percentage of disability which the injured workman or plaintiff has suffered. A few weeks or a few months under the benign treatment of the optimist and the patient is as good as ever, ready to take his place in industry as if he had never been hurt. You can well understand why the insurance companies and corporations love the optimists.

It is not up to me to sermonize on this point. If I told you that I believe that the plaintiff's doctors, or shall I say the pessimists, are the more realistic, you might well conclude that I prefer to believe the patient. As a matter of fact, we are dealing with a human equation wherein we must recognize the fact that various people have different thresholds of pain, some are more persistent than others in overcoming disability, and some may endure physical defects and handicaps without yielding to the temptation to complain.

On the other hand, I am satisfied by my experience that there is an element of neurosis in almost every case of trauma and disease and that the physician must treat the mind as he treats the body to the end that after he has cured a physical defect he does not create a mental aberration.

I am happy to say that I have a healthy respect and admiration for the members of your fine profession in this community, the pessimists and optimists alike, as I have found practically all of you with whom I have come in contact faithful and loyal to your profession and your patients, friendly, and helpful, doing an excellent job in upholding the finest tradition of your great profession.

Temperance and labor are the two best physicians of man: labor sharpens the appetite and temperance prevents him from indulging to excess. —*Rousseau*

THE MONTH IN WASHINGTON

Editor's Note. The following summary of Washington news was prepared by the Washington office of the A.M.A. for distribution to state and regional medical journals.

The controversial health reinsurance issue has come back into prominence, and under conditions that make the whole question about as complicated as it can get. The bill would have the federal government underwrite voluntary health insurance plans if they agree to experiment with risks not usually covered.

Although this measure is a major part of President Eisenhower's health program, it became bogged down in the House Interstate and Foreign Commerce Committee when widespread opposition developed. Then the committee chairman, Rep. Charles E. Wolverton (R.-N.J.), turned to one of his favorite subjects, a plan for federal guarantee of private loans to health facilities for construction and equipment. This bill, however, was not supported by the administration.

In an effort to placate the opposition, Mr. Wolverton offered to eliminate a number of objectionable features from the mortgage guarantee bill. At the same time there were reports that he proposed to merge this bill with the administration-supported reinsurance bill. Meanwhile, Henry J. Kaiser made two special trips to Washington to help out his friend, Mr. Wolverton, by putting his weight behind the mortgage loan idea. That was not surprising, inasmuch as Mr. Kaiser had helped to draw up the bill, which would greatly benefit health centers such as those started on the west coast by the Kaiser Foundation.

Mr. Kaiser, saying he was producing a film to promote the mortgage loan plan, went to the unusual extent of making a direct appeal to Washington news correspondents to write favorable copy about the bill.

While these Wolverton-Kaiser maneuverings were taking place on the mortgage bill, it became apparent that President Eisenhower was not ready to abandon the reinsurance idea. He called a number of executives of major life insurance companies to the White House to try to impress them with the merits of reinsurance and in other ways indicated he still wanted to see the bill passed this session. Secretary Hobby, whose original testimony for reinsurance had been restrained, also joined in the last-minute campaign. But it appeared the tangle might be too complicated even for Mr. Eisenhower to unravel before adjournment.

Most other parts of the Eisenhower health program were moving through Congress, even though some were off schedule. (Of the major bills, A.M.A. opposes only reinsurance.) Legislation to expand the Hill-Burton hospital construction program cleared what might have been a serious obstacle when it was reported out by the Senate committee. Compared with the House bill, the Senate bill gave more discretion to state health authorities in use of funds for constructing facilities for the chronically ill, for nursing homes, and for health centers. However, the Senate would require that funds earmarked for rehabilitation centers be used for the stated purpose. The Senate also would rule out the possibility of U.S. grants to centers devoted solely to treatment. Unless the facility could qualify as a diagnostic center, or a diagnostic-treatment center, it could not be eligible under the Senate bill. This safeguard was not in the House bill.

Of the remaining legislation of interest to the medical profession, the status at this writing was about as follows:

The doctor draft amendment, to strengthen Defense Department's hand in dealing with physicians who might be security risks, had passed the Senate, been reported by the House committee, and was almost a law. Also about to be enacted was a provision liberalizing medical expense deductions from taxable income. The long-dormant bill to transfer responsibility for Indians' health matters from the Indian Bureau in Interior Department to Public Health Service in the Department of Health, Education, and Welfare was pointed toward enactment, but might possibly be held up by objections of Senators from a few western states. The Interior Department had dropped its original objection.

The House-passed social security bill, with the compulsory coverage of physicians eliminated, was before the Senate Finance Committee, where anything could happen. Two bills of medical interest already had been passed by both houses and signed into law. One prohibits the shipment of fireworks into a state where fireworks are illegal, and the other relieves Army medical officers of the technical responsibility for supervising preparation of food.

A reassuring note was sounded by President Eisenhower when he forwarded to Congress the controversial International Labor Organization convention on minimum standards of social security with a recommendation that it not be ratified. His message said most of the points—including a suggestion for socialized medicine—were not proper subjects for the Congress to deal with.

Physicians visiting Chicago are invited to tour the A.M.A. building at any time. Records show that the number of physicians and medical society executives taking the tour during 1953 more than doubled the number for the previous year.

PRESIDENT'S PAGE

DEAR DOCTOR:

During the hot summer many of us look forward to a restful vacation. Some turn to fishing, others relax on the beach, and still others might find a trip to the mountains most restful. While it goes without saying that one is entitled to a certain amount of leisure, it is most heartening to see the program of The Kansas Medical Society go forward.

The month of July is filled with meetings of our various standing committees earnestly working to carry out unfinished business of the preceding year, or introducing new projects in an effort to further medicine professionally and from a public relations standpoint.

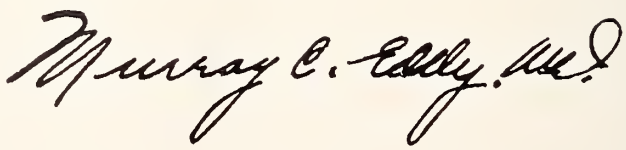
It has been necessary to appoint special committees to carry out programs not specifically designated for a standing committee. Your response in this regard has been most gratifying.

The doctors in Reno and surrounding counties who have already had several meetings to discuss and work out a program for our 1955 annual session are holding another such meeting this month.

While your officers are most willing to do what they can to further Society activities, the success of our program is determined by the continued effort of individual members. Committee chairmen will introduce to their committees worth while suggestions from any member of the Society.

May I thank you on behalf of your officers for your excellent co-operation to date and encourage your continued participation in the year ahead.

Yours sincerely,



EDITORIAL COMMENT

Federal Health Programs

PART I

During the fiscal year closing June 30, 1954, the federal government will have appropriated some $1,775,882,197 for health activities. This is more than 1¾ billion dollars, and in the final accounting the figure might even surpass that amount.

According to an analysis prepared by the A.M.A. Washington office, the federal government operates or participates in more than 60 health, medical, and related programs which are operated by 19 different departments, agencies, or commissions. Not all these agencies can give accurate fiscal expenditures until the year is concluded, but the figures are presumed to be reasonably correct.

Some of these programs are operated directly at the federal level, but most are largely distributed to the different states upon some pre-determined formula. How Kansas uses these and other funds will be recorded in future articles. There will be no attempt made to criticize any program mentioned. This series is intended to be explanatory. Statistics cannot be avoided if this story is to be told.

The federal government is in the business of health care. This series of articles will attempt to describe these programs. The physician interested in this subject may then determine for himself whether or not he approves of what is being done.

The Department of Health, Education and Welfare spent a total of $340,553,000 during this fiscal year, or about one-fifth of the total federal health expenditure. All the following are administered under this one department.

The Office of Vocational Rehabilitation used $23,655,500, of which $655,500 represents federal administrative costs. The remainder was given to the states on a dollar for dollar matching basis to give medical care, hospitalization, prosthetic appliances, vocational training, etc., to handicapped persons.

The Food and Drug Administration spent $6,250,000 to enforce laws designed to protect the public against adulteration and the misbranding of foods, drugs, and cosmetics.

The Children's Bureau spent $31,525,000 for crippled children and maternal and child health programs. Federal administration expenses are $1,525,000 so 30 million was spent to match state appropriations for such programs.

The Bureau of Public Assistance spent $50,000,000 for medical and health programs. Federal administration is about $1,550,000. This is a complicated formula, and a complete description would require more space than its value warrants. The briefest account is to say that the federal government spent $1,341,550,000 for public assistance and that about 4 per cent of this total was for health care. This was paid either directly to the vendor or to the client in the four assistance categories—old age, blind, permanently disabled, and dependent children. Included in the above figure is about 50 per cent of the administrative expenses within the various states.

Also included under this major department is the United States Public Health Service which operates all of the following medical programs. These are briefly recorded as follows:

The Office of the Surgeon General spent $2,900,000 for administrative expenses, surveys, international health activities, training programs, etc.

Tuberculosis Control spent $6,000,000, of which something more than two-thirds was given to states for case finding, treatment, and follow-up services on a basis of two federal dollars for each state dollar spent. The remainder was used by the U.S.P.H.S. to finance direct activities including drug evaluations and BCG vaccinations.

Venereal Disease Control spent 5 million dollars, of which just under 2 million was given to the states for treatment and control on a two-to-one basis, the federal grant being twice the state expenditure. Something over 2 million dollars was used for training programs or to pay salaries of federal employees assigned to the states, and the remainder was spent directly by the U.S.P.H.S. for tests and demonstrations.

Assistance to the states accounted for $13,250,000, of which just over 3 million dollars is spent directly by the U.S.P.H.S. for consultant services, training programs, etc. The various states received $10,135,000 on a basis of two federal dollars for each state dollar for general public health purposes.

Communicable Disease Control used 5 million dollars, all of which was spent by the Communicable Disease Center at Atlanta, Georgia, for studies in epidemiology, laboratory services, and special services to the states in case of epidemics.

Engineering, Sanitation, and Industrial Hygiene spent $3,162,500, of which $87,000 was for administrative expenses. Water supply and pollution control used $903,500; studies of radioactive substances $275,000; sanitation, milk and restaurant inspection, garbage disposal, etc., $799,000; health hazards of manufacturing and mining $550,000, and environmental research $547,500.

Alaska received $1,082,000 for public health services and to finance the Arctic Health Research Center at Anchorage.

Hospitals and Medical Care used just over 33 million dollars for the operation of hospitals and health

services for American seamen, Coast Guard and Public Health Service personnel and their dependents, for federal employees injured at work, for leprosy patients, and for narcotic addicts.

The Foreign Quarantine Service spent $2,900,000 to operate 250 quarantine stations located on the borders of the United States. It also operates about 25 medical stations in foreign countries where aliens who seek to enter this country may be examined. And, finally, this service inspects all seagoing vessels and aircraft entering the United States.

The various National Institutes of Health spent $71,153,000, of which the National Cancer Institute received $20,237,000. About two-thirds of this latter was distributed as research grants, just over 2 million dollars was allocated to the states for cancer control programs, and the remainder represents the cost of operating the Bethesda (Maryland) Clinical Center.

The Mental Health Institute received $12,095,000, of which 60 per cent went to individual investigators working on research programs. Two and one-quarter million dollars was allocated to the states, and the rest was spent at the Bethesda Clinical Center.

The National Heart Institute received $15,168,000 and diverted this money according to the same formula as above. Similar also are the Dental Health Institute, $1,740,000; the Microbiological Institute, $5,738,000; the Institute for Neurological Diseases and Blindness, $4,500,000; the Institute of Arthritis and Metabolic Diseases, $7,000,000, and the National Institutes of Health, General Funds, $4,675,000, except the last named fund is almost all used for grants and some of the others do not distribute money for state uses.

Also under the United States Public Health Service is the hospital construction program which is listed under three divisions, obligational cash for new construction under the Hill-Burton Act, 65 million dollars; liquidational cash to complete previously authorized projects, $19,700,000, and administrative expenses $875,000. This program also operates under a complicated formula and with local recommendations, but in general it represents aid for hospital construction whereby the federal government contributes from one-third to two-thirds of the total cost, depending upon the per capita wealth of the state. Since 1946, when this program was started, federal funds have partially financed 2,100 projects with 102,000 hospital beds and in addition aided in the construction of 400 health centers.

This, then, is a summary of health expenditures through the Department of Health, Education and Welfare—some 340 million dollars out of a total of 1¾ billion spent for health by the federal government. The discussion will be continued next month describing such agencies as the Veterans Administration, the State Department, and others.

Malpractice Insurance

Some considerable confusion has arisen over the subject of professional risk insurance for physicians. A generalized national increase in malpractice actions, together with the apparent trend of higher judgments awarded by juries, has caused policy changes among insurance companies.

The majority have organized to agree upon a standard policy, uniform acquisition practices, and equal premium rates. In brief, these companies set rates for each state according to the experience within the area. As may be expected, the cost in coastal and industrial regions is considerably greater than for Kansas. Policy types vary according to specialty practice, and rates differ in certain fields. There are some uniform exclusions such as spinal anesthesia and shock therapy written into all of these policies. The physician is also required to purchase certain other forms of liability protection such as automobile or fire from the same company. However, he may buy practically unlimited protection.

A very few companies did not join this group and continue with their own individual insurance customs. One company issues a policy notably less expensive but operates upon a strict theory of low coverage with no exclusions and careful attention to defense. Both theories have found support in Kansas, and it is not here intended to argue the point. Essential differences should be understood, however, so this primer is prepared to represent an impartial review upon this subject.

The first comment is that every physician should carry some form of insurance protection for each day he is in practice. For the great majority this will never be needed, but its service is not only protection against carelessness but principally to cover suits for situations that are completely beyond the physician's control.

Some members continue their mistaken belief that The Kansas Medical Society provides insurance protection. This is not true and should be understood. The Society elects a Defense Board of three physicians who will assist, if the case appears defensible, any member requesting such aid. The attorney for The Kansas Medical Society may be provided to aid such member, but the cost of his services will be paid by the defendant. The Society will aid in defense if it appears proper, but the Society will not pay judgments or court costs or attorney fees arising out of any malpractice suit.

Second, the individual physician should determine

for himself how much insurance he wishes to have and what company he proposes to select for this protection. Physicians who are entering practice might profit from consulting with established physicians regarding local custom and experience. Philosophy upon this subject will vary. Some will want high limits to protect their personal property against loss by court action. Others will argue that careful defense and low limits will serve to reduce the size of judgments. This decision should be based upon personal preference.

One theory holds that high limits invite suits and subsequent high judgments. The other declares low limits to leave the doctor vulnerable to personal loss. One holds that careful attention to details and thorough defense preparation practically eliminate this danger and argues that experience proves this to be true. The other theory simply denies that statement and speaks of hazards that accompany less than full financial security. One says they have proved the soundness of their position, the other asks what about tomorrow. Low limit advocates tend to credit those holding the other theory with a considerable responsibility for the present situation, while they in turn argue that the present situation presents additional proof for the need of carrying high limit protection.

One theory holds that the plaintiff is more interested in suing the impersonal insurance company than the doctor, but that also is argued against. High judgments and liberal out-of-court settlements are contagious. One such action frequently results in other cases being filed from the area, but again the argument can be turned both ways. So, again, this discussion is not intended for advice on the question of amount. The important thing is that the physician carry insurance; how much and with which company shall be decided by himself.

Regardless of that choice, there are many things a physician may do to prevent his becoming involved in legal action of this type. Even though all may be summarized by saying that careful attention to the practice of sound, scientific medicine and conscientious adherence to the principles of ethics represent the best possible protection, perhaps a few points should still be mentioned. The JOURNAL is indebted to the Medical Protective Company of Fort Wayne, Indiana, for permission to reprint this list of 30 suggestions which every doctor should follow.

1. Keep accurate records permanently. Suits sometimes arise scores of years after the rendering of services.

2. Keep abreast with the average standards of his field of practice in his locality. This the Law requires him to observe.

3. Avoid criticism of the work of other doctors. To

SERVICE SEPARATIONS

As a service to physicians and communities in this state desiring additional medical personnel, the Journal of the Kansas Medical Society will publish in this column each month the names of medical officers who will shortly be separated from the armed forces. These are men who volunteered from Kansas, and many of them will probably be interested in finding locations in this state. Anyone interested in contacting these physicians may write to the address here given.

Theodore L. Batchelder, M.D.
Garden City, Kansas

Albert I. Decker, Jr., M.D.
826 Alabama Street
Lawrence, Kansas

Jack A. Dunagin, M.D.
1266 Collins
Topeka, Kansas

Lyle Harris Edelblute, M.D.
1210 Thurston Street
Manhattan, Kansas

Edgar F. Ewen, M.D.
1022 East 5th
Cherryvale, Kansas

Simon E. Hershorn, M.D.
Wesley Hospital
Wichita, Kansas

Everett E. Howard, M.D.
1708 North Grand
Pittsburg, Kansas

J. D. Kabler, M.D.
1506 Parker Avenue
Wichita, Kansas

J. Gilleran Kendrick, M.D.
4224 East English
Wichita, Kansas

Francis X. Lenski, Jr., M.D.
703 South Cottonwood
Iola, Kansas

Stanley R. McEwen, M.D.
447 North Belmont
Wichita, Kansas

Alexander H. Milne, M.D.
2420 West 25th Street
Topeka, Kansas

Owen C. Peck, M.D.
2000 Oakland
Kansas City, Kansas

Ralph B. Reauwe, M.D.
524 West 7th
Junction City, Kansas

Julian W. Reed, M.D.
703 North Pine
Beloit, Kansas

Robert N. Shears, M.D.
15 Carlton Road
Hutchinson, Kansas

Grant M. Stevens, M.D.
305 Crescent Boulevard
Hutchinson, Kansas

Charles E. Wilder, M.D.
Winter VA Hospital
Topeka, Kansas

do otherwise not only invites retaliation but also weakens his own defenses.

4. Avoid admissions against his own interest on services he himself has rendered or anyone else has rendered in his behalf. Any such admissions will probably be used against him.

5. Learn and observe his legal duty to his patient.

6. Use the x-ray as a diagnostic or confirmatory aid where that is established practice.

7. Guarantee no results. Such guarantees are not only unethical but uninsurable.

8. Co-operate with the patient in securing aid of consultants. These strengthen defenses.

9. Give proper notice of termination of services so as to avoid charges of abandonment.

10. Secure proper written consent for operation in every instance. Without such evidence he may be charged with assault.

11. Keep his relations with his patient strictly confidential.

12. Avoid divulging that he has professional liability coverage.

13. Refrain from unapproved or experimental procedures.

14. Be courteous and tactful in relations with patient.

15. Advise patient of any absence from practice and recommend or make available a competent substitute.

16. Make certain that proper instructions are given with prescriptions or in the care of the patient.

17. Secure proper authorization for any autopsy in which he participates.

18. Have, whenever possible, unprejudiced witnesses of his relations with the patient, especially in the case of female patients.

19. Have a clear understanding relative to his fees before services are rendered, especially if such fees are of sizable amounts.

20. Postpone, whenever possible, any suit for fees until after the statute of limitations on malpractice suits has expired.

21. Secure legal advice before attending a coroner's inquest as a witness.

22. Secure legal advice on how to make his personal assets less vulnerable to damage suit attacks.

23. Avoid sterilization procedures except where medically indicated.

24. Avoid telephonic prescriptions without proper verification.

25. Keep all equipment in proper working order.

26. Avoid fields in which he is not properly qualified.

27. Keep all promised appointments.

28. Give proper instructions to assistants and make certain that they are observed.

29. Maintain due frequency of visits for proper care of patient.

30. Exercise due care at all times, especially in the labelling of bottles or other containers, in the use of mouth gags, in the close observation of casts, in periodic check of needles and other equipment, in requiring personal examination for commitment proceedings, and similar procedures.

Physician Placement

Kansas physicians are aware of the success of the Kansas Plan in helping place doctors in communities where medical care is urgently needed. At present, 68 communities in Kansas with populations under 500 have medical doctors. More than 150 towns with populations under 1,000 have physicians in their communities.

The work has not been completed, however, and the Executive Office is continuing its efforts to advise inquiring physicians where such situations exist.

It becomes necessary periodically to revise the locations list as various communities obtain the services of a physician and others are placed on the critical list. In some instances a serious situation may exist that is not known to the Executive Office, or the reverse may be true when a doctor locates in a community and the office is not informed of that fact.

In order to revise the list at this time, the help of every physician is needed to inform the Executive Office of places requiring the services of additional medical personnel. If the list is kept accurately, the Kansas Plan will continue to produce results and will help Kansas retain its outstanding record among the 48 states in this endeavor.

Study on Ulcers

Ulcers, caught in time, do not necessarily represent an unusual hazard to longevity, according to a recent release from the Institute of Life Insurance. That fact, along with others, resulted from a study of 15 years' mortality experience among 725,000 life insurance policyholders.

Of about 45,000 cases with a record of ulcers studied by the Society of Actuaries, 96 per cent of them men, those who had been medically treated, without an operation and without having had a hemorrhage, were found to have had a relatively favorable mortality experience during the 15 years covered. Three-fourth of the total ulcer cases were duodenal, the balance being gastric.

Another section of the study, covering persons with gall bladder disorders, indicated that such impairments are much more frequent among women than among men.

The Complications of Polycythemia Vera

Curtis A. Nystrom, M.D.
Kansas City, Missouri

Polycythemia vera, first described by Vaquez in 1892 and clearly defined by Osler in 1903, has presented several puzzling features to medical investigators.

Polycythemia vera is a comparatively rare disease, characterized by a great increase in circulating blood of the red blood cells and hemoglobin with a color index below unity, hematocrit elevated to 70 per cent and total blood volume increased. In present nomenclature it is called polycythemia hypervolemia.[1] Synonyms are erythremia, splenomegalic polycythemia, Vasquez's disease, Osler's disease, polycythemia rubra, polycythemia with chronic cyanosis, myelopathic polycythemia (Weber), erythrocytosis megalosplenica (Senator), and cryptogenic polycythemia (R. C. Cabot).

The etiology is unknown, but it frequently develops between the ages of 40 and 50, although it occurs at all ages. Records of 6 hospitals indicate that it is higher in Jews of eastern European descent.[4] (Forty-eight per cent of 134 cases—admission rate when average in these hospitals of this race was 10 per cent.) Meakins believes that there is no sex predominance, but Sturgis lists the ratio in males to females as 2:1. Hypotheses as to the etiology include anoxia of the bone marrow,[22, 16, 28] fibrotic narrowing of the vessels,[9, 12] neoplastic changes in the stem cells of the bone marrow,[15, 22, 27] hypersecretion of the intrinsic erythropoietic factor,[21] and hypersensitivity of the carotid sinus and the aortic nerves.[3]

Sodeman[29] feels that anoxia is the basis of polycythemia vera, but he does not feel that every type of polycythemia vera is due to the same type of anoxia. The type described as being associated with hypertension without splenomegaly is caused by stagnant anoxia. Stagnant anoxia is caused by either structural alteration or vasoconstrictor influences. Patients with "splenomegalic polycythemia vera" have a polycythemia which may be viewed as secondary to an underlying process often leukemic or leukemoid in character. This type is due to a competitive anoxia. Initially, competition for available oxygen by numerous immature white cells or later progressive fibrosis and osteosclerosis of the bone marrow with consequent local disturbances to blood flow may result in local anoxia with consequent stimulation of erythropoiesis. This, Sodeman feels, will even account for secondary polycythemias so that the physiologic mechanism is anoxia of the erythropoietic cells. It is erroneously regarded as a primary neoplastic disease of the red cell only because of its frequent and close association with such processes in leukemia and especially in so-called non-leukemic myelosis.

The polycythemic phase may be accompanied by leukocythemia or thrombocythemia or both. Any and all combinations are possible. When it is realized that exhaustion of one element may occur while another begins or continues to hyperfunction, one can more readily understand the wide variation in hematologic pictures presented during the slow progress of this disease. It should be remembered that after therapy some patients have normal values for a number of years, and then the red blood count begins to return to its former high reading, but the hemoglobin may or may not increase. This will cause the stained smear and the color index to present the picture of severe hypochromic anemia.

Diagnosis is generally made by the following criteria:[4] (1) polycythemia which was distinct and persistent and which returned after cessation of therapy, (2) splenomegaly, (3) typical red cyanotic appearance of patient, and (4) absence of any other distinct lesion, such as pulmonary or cardiac, to explain the polycythemia. Other signs and symptoms are headache, vertigo, fainting, and fullness in the head, and in the fundi the veins are much engorged and hemorrhages are frequently seen. There may be disturbed vision and sometimes hemianopsia or diplopia.

Analogous to these symptoms there may be paresthesias and peripheral paralyses. These are usually accounted for by focal hemorrhages or thrombosis in the central nervous system. Other symptoms of central nervous system origin may be periods of slow cerebration, amnesia, irritability, and tinnitus. There is often weakness.

Abdominal pain and cramps are complained of, the former particularly over the splenic enlargement, which may be accounted for by splenic infarctions, and the latter by gastrointestinal infarctions which may lead to hematemesis and melena.[10, 26] Precordial pain indicates myocardial infarction, which is also suggested by the electrocardiogram. The most important signs are cyanosis, a plethoric facies, sple-

This is one of 11 theses, written by fourth year students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group judged to be the best by the faculty at the school.

Thesis written while the author was a fourth year student. Dr. Nystrom is now interning at St. Mary's Hospital, Kansas City, Missouri.

nomegaly—sometimes assuming enormous proportions. There may be hypercalcemia and a hypochloremia. The basal metabolism rate is elevated by 15-25 per cent or higher. Blood pressure is elevated, and hypertension might be present.

The red blood count is elevated to 7-12 million with a hemoglobin of 150-160 per cent. The color index is below unity. There is possible irregularity in the red blood cells in shape and size; there is always evidence of immaturity as shown by polychromatophilia and normoblastic forms. Usually a leukocytosis is due to increase in granulocytes, but abnormal varieties are seldom found. The platelets are increased.[1] In about 75 per cent of the cases the spleen is enlarged; the liver is enlarged in about 50 per cent. X-ray reveals increased vascular markings in the lung fields. There is frequent bleeding from the nose and gums; this is not due to a tendency to bleeding but is the result of congestion of the vascular bed.[26] Total blood volume is increased.[5, 9]

There are a number of complications listed that occur in polycythemia vera:

Cerebral thrombosis	16.5%
Suspected brain tumor	4.9%
Choked disk	2.4%
Herpes	0.6%
Combined sclerosis	0.6%
Gout (possibly a complication of a pre-existing condition)[6]	4.7%

Strobel classifies polycythemia vera into types according to the degree of leukocytic reaction in the peripheral blood, as follows:[28]

Type I—Simple polycythemia vera, characterized by normal or slightly increased leukocyte count or by a slight increase in the per cent of polymorphonuclear neutrophils.

Type II—Polycythemia vera with mature leukocytosis (more than 15,000 mature leukocytes per cubic millimeter of blood).

Type III—Leukemoid polycythemia vera, characterized by leukocytosis and the presence of immature leukocytes.

(Transition occurs from one type to another in time and following treatment.)

	82 pts. Type I	30 pts. Type II	30 pts. Type III
No change	75%		10%
To Type I			43.3%
To Type II			
To Type III	24.4%	23.3%	20%
Acute leukemia	no-breakdown		

All patients had a five-year follow-up, but 25 of Type I had a 10-year follow-up.

Using this classification, it was seen that splenomegaly and hyperuricemia was about equal in Type I and Type II but was marked in Type III. Gout occurred almost twice as frequently in Type II as in Type I. The serum uric acid was increased in 54 per cent of Type III with 12 of the 14 cases having clinical gout.

Complications which have their manifestation on the skin include hemorrhages with resulting brownish pigmentation. There may be purpuric spots and even hematomata. Eczema and dryness are not unusual. Acne-form, urticarial eruption with rose-red coloration over firm, irregular round or oval nodules, very tender to touch and scattered over the trunk, have been reported.[8]

The appearance of the nail-fold capillaries as compared with normal has led Brown and Giffin[9] to classify vascular changes in polycythemia vera as chiefly concerned with (1) readjustment of the vascular bed to the augmented blood volume; (2) changes in vessel walls, probably due to strain, metabolic disturbances, and various other factors; (3) vascular plugging, probably due to thrombotic processes as a result of increased viscosity and changes in vessel wall and the coagulation factors of the blood; and (4) changes in the erythrogenic tissue.

In the first stages of the disease the accommodation of the blood vessels occurs in the large veins and in the more vascular organs, especially the spleen. As the condition progresses the smaller veins participate in the engorgement, as can be observed in the peripheral and the retinal veins. With more advancement comes the plethoric facies and the bluish-red mucous membranes. Probably the symptoms of polycythemia vera are not produced until the engorgement progresses to the smallest vessels.

Cardiac hypertrophy is often seen following increased pulmonary congestion and increased viscosity of the blood. Vascular diseases include venous thrombosis, varicosities, and phlebitis.[8] Many patients have episodes of phlebitis years prior to development of other symptoms of polycythemia vera.[22]

Vascular symptoms which occur early in patients who later go on to develop or have early polycythemia vera are due to vascular occlusions in the brain, in the coronary arteries, hepatic veins and arteries, and veins of the extremities. There are also circulatory disturbances without clinical evidence of demonstrable occlusion. Since a circulatory disturbance in the limbs, especially the lower limbs, is often one of the earliest subjective symptoms of polycythemia vera and presents much the same picture as thromboangiitis obliterans or arteriosclerotic occlusion of the vessels of the legs, all patients with obscure vascular disturbances should be examined for possible polycythemia vera.[12]

There are vasomotor disturbances of the dilator type suggesting erythromelagia, with features like those of Raynaud's disease. This is possibly explained by the microscopic studies done on the blood vessels

in normal patients, in patients with polycythemia vera, and in those with thromboangiitis obliterans. It was found that the polycythemia vera and thromboangiitis obliteran sections were much alike and showed thickening of the capillary walls with increase in fibrous tissue. They showed marked subintimal and adventitial fibrosis of the arteries and arterioles. Some of the fibrosis extended into and through the media.[4]

Failure to keep the nervous system manifestation of polycythemia vera in mind will lead to diagnostic mistakes. Many of these patients have symptoms referable to the nervous system which may be extensive and sometimes confused with primary neurological conditions or surgically relievable tumors or masses.[7, 12] One must not forget that many diseases are associated with polycythemia vera, and these include those of neurologic nature. A list of many neurologic symptoms and their frequency follows:[7]

Total cases	56
Headache	33
Vertigo	30
General weakness	15
Easy fatigability	13
Fullness in the head Paresthesia Mental impairment	11
Visual disturbances Pain Nausea and emesis	9
Nervousness and irritability	8
Emotionalism	5
Sleep disturbances General pruritus Tinnitus	4

Various symptoms observed in reference to the eye are thought to be caused either by engorgement or by small thromboses. Changes in the fundus are more or less those called cyanosis retinae by Liebreich. The color of the fundus is darker red, the veins are greatly enlarged and tortuous. Many veins are visible because even the smallest veins are filled with blood. Two patients were directly observed to have spasm of the central retinal artery. The veins became greatly dilated and tortuous. The arteries were so free of blood that they appeared as broad white stripes. During these and other possibly similar episodes, the patient's vision deteriorated rapidly until generally only handwaving was visible. With recovery from the spasm, blood could be seen flowing back into the arteries with a resultant gradual, but not complete, return of vision. There was also an example of the formation of new blood vessels and retinal hemorrhages.[13] Meniere's syndrome has been observed and is believed to be caused by distension and engorgement of the vessels of the middle ear.[8] Papilledema is seen as a complication.[16]

Vascular lesions of the brain are the most serious complication of this disease and are a common cause of death. Nervous symptoms are apprehension, nervousness, excitability, irritability, hypochondriasis, disturbed mentality, insanity, delirium, insomnia, muscular atrophy, numbness, epileptiform attacks, muscular twitchings, shivering, tremor, loss of consciousness, aphasia, paralysis, and choreiform movements.[14] Pollack[14] had a case which he thought was due to a lesion in the basal ganglion. This was thought to be due to a definite pathological condition in the brain such as hemorrhage or thrombosis, for choreiform movements later disappeared, but erythremia persisted. (Some authors prefer to use the term erythremia to polycythemia vera.) Any pathological condition in the brain caused by hemorrhage or thrombosis can be produced by polycythemia vera.

Disturbed circulation and abdominal plethora cause many symptoms in the abdomen, some of which must be thought of in an acute abdomen.[11] The patient may have a fullness of the stomach, thirst, gas pains, belching, and chronic constipation. There is frequently pain and tenderness over the spleen, occasionally pain into the left shoulder or down the left side of the body and into the left leg. Hemorrhage, often massive, is an important complication. Thrombosis, when in the mesenteric veins and arteries, may be mistaken for peritonitis.

There are many cases of erythremia listed in the literature. A typical patient entered the Massachusetts General Hospital with chief complaints of dyspnea on exertion, edema, abdominal cramps, and melena. She had had an episode of left leg swelling and pain 17 years before admission. She was admitted to the hospital at a prior time following an acute myocardial infection with acute pulmonary edema and lower abdominal cramps. The patient was treated for her myocardial infarction at that time and was dismissed. The second admission was two years later, and at this time she entered with massive pleural effusion, ascites, pulmonary edema, right- and left-sided failure, hypotension, nausea, and vomiting. The melena was due to mesenteric artery thrombosis.[18]

Friedman finds that the blood of patients with gastric ulcer is generally anemic and with duodenal ulcer it is polycythemic and rarely anemic.[20] He believes that the ulcers found in polycythemia vera patients are due to adrenalin,[19] but the occurrence of thrombosis of a gastric vessel is suggested. The occurrence was 8 per cent in 143 cases, and the incidence of ulcer in 200 controls was 2 per cent.[21]

A 69-year-old female entered the Massachusetts General Hospital with the chief complaint of an abdominal mass which had been increasing in size for seven years. The mass when first detected was the size of a walnut, located in the left lower quadrant and without associated symptoms. Three years prior to admission it had markedly increased in size. The patient now had increased urinary frequency, nocturia, and anorexia. At the same time she began to

notice a tendency to bruise easily. She had some discomfort when erect and required medication to sleep. With increase in the mass size, she had developed moderate dyspnea and some palpitation on exertion.

She had lost 30 pounds in the last three years without nausea, vomiting, or change in bowel habits. No vaginal bleeding occurred since menopause at age of 53.

Physical examination revealed an emaciated female with bronze pigmentation of exposed skin areas and multiple ecchymotic spots over the arms and upper trunk. Her color was high but not violaceous. There were a few telangiectases present over both cheeks and right ear. The heart was enlarged to percussion, and there were irregularly frequent extrasystoles. The second aortic sound was louder than the second pulmonic sound. The mass filled the abdomen except the right upper quadrant. It was not easily movable, but it was not attached to the abdominal wall. A definite notch was palpable along its right border below the level of the umbilicus. It was not palpable rectally, and pelvic examination was within normal limits. The liver was down four fingerbreadths below the costal margin. Distention of superficial veins in the neck and left leg was seen.

Laboratory work revealed a white blood count of 23,850 with 87 per cent neutrophils. Granulocytes showed a shift to the right. The red blood count was 8.26 million with hemoglobin of 18.5 grams. The hematocrit was 76 per cent. The red blood count showed considerable variation in size and shape with some achromia, many cells showing polychromatophilia. Reticulocyte count was 7 per cent with occasional normoblasts. Platelets were diminished, and bleeding time was 1.5 minutes while clotting time was 11 minutes. Stool guaiac examination was negative. Hepatogram revealed some hepatic impairment. A barium enema showed downward displacement of the major part of the colon by an extrinsic mass. Basal metabolism rate on two occasions was plus 69 and plus 59.

On the seventh hospital day a sternal bone-marrow aspiration was done. The myeloid-erythroid ratio was 9:1 with a marked shift of myeloid maturation to the right. No blast forms were seen, and there were rare premyelocytes. Megakaryocytes were plentiful. No plasma cells were seen.

Following a sternal bone-marrow biopsy one week later, which revealed much the same blood picture in the bone-marrow, the patient developed a painful subpectoral hematoma in the site of the biopsy. This was surgically evacuated following clotting, but she had an exacerbation three days later and died two days following this second episode.

It was thought necessary in this case to rule out ovarian tumor since the mass was first noticed in the left lower quadrant, but without vaginal bleeding and with a normal pelvic examination this was not thought likely. Leukemia was thought of as she had such a high basal metabolism rate, but all of the white blood cells were mature. Few patients live this long with the disease, so it is not known whether or not the basal metabolism rate is normal. It could be accounted for by the large amount of erythropoietic tissue found. X-ray showed multiple vertebral fractures involving the lower thoracic and upper lumbar segments, also a rib fracture on the right side, with all bones decalcified (normal at this age). Her thin bones were also due to the fact that they were full of actively regenerating blood, and the trabeculae had been hollowed out by this long, slow disease. At autopsy there was also found blockage of some of the pyramids of the kidneys by urates.[23]

While hepatic enlargement is common in polycythemia vera, the presence of marked enlargement frequently signifies an important complication. Phenlyhydrazine jaundice, leukemic transformation, and myocardial decompensation are not uncommon causes of considerable hepatic enlargement and are readily detectable. Cirrhosis of the liver and thrombosis of the hepatic and portal veins remain to explain a small number of cases, and the condition is usually diagnosed at autopsy.[23]

Study of the collected reports of cases discloses such points of diagnosis usefulness as sudden enlargement of the liver, rapid accumulation of ascites, resistance of ascites to mercurial diuretics, frequent terminal jaundice, and a marked abnormal plasma cholesterol partition in thrombosis of the hepatic veins, occasional decrease in the size of the liver and effective diuretic measures in ascites of cirrhosis, and the infrequency of ascites, hepatic enlargement, and jaundice in the thrombosis of the portal vein.[23, 24]

A basic change in all five cases studied by Newman, Feltman, and Devlin was an increase in the maximum breathing capacity after phlebotomy. This is believed to be due to an increase in the elasticity and a decrease in the viscous resistance of the lung tissue to changes in shape, and, when present, to a decrease in the anoxemia. The increase in maximum breathing capacity was probably due primarily to a decrease in blood viscosity and to some extent a decrease in total blood volume. Three of their patients revealed papilledema, and one had painless gangrenous changes at the tips of the distal phalanges of the four medial fingers of the left hand.[16]

Barker[17] feels that cases in which both polycythemia and chronic pulmonary disease are present should be divided into four groups:

Group I—Primary pulmonary disease with compensatory polycythemia—here blood volume is not increased, and the treatment of the polycythemia would be detrimental to the patient.

Group II—Primary pulmonary disease in which

the polycythemic response has overcompensated and there is an increased total blood volume.

Group III—Polycythemia vera with complicating but unassociated pulmonary disease.

Group IV—Those cases of polycythemia vera causing or predisposing to pulmonary disease. Patients with polycythemia vera have dypsnea on exertion, vital capacity is reduced, and there is pulmonary congestion. These patients are easily affected with an upper respiratory infection. This causes a vicious cycle. The last three groups would benefit from treatment of the polycythemia. Hoarseness is not unusual, for the vocal cords are excessively red.[8]

The only effect that polycythemia vera has on the kidney is that of hemorrhage in the capsule,[25] accumulation of urates in the pyramids,[22] and displacement of the kidney with angulation of the ureter by the enlarging spleen.[10]

There is excessive nucleoprotein breakdown with excretion of considerable amounts of porphyrins[2] and uric acid in the course of active erythropoiesis.[10, 11, 22] This high concentration of uric acids either causes gout or is the overload which brings out an impending case.[29]

When polycythemia vera goes on for years, the condition terminates in many other conditions, such as the subsequent development of myeloma. This is thought to be due to neoplastic changes in the stem cells of the bone marrow. In these cases, the stem cell may first have differentiated into the red cell series to cause the elevated red blood count which later decreased when the stem cell differentiated into myeloma cells.[15] The course is an irregular one, and death is usually due to vascular accidents or a failing circulation. Great enlargement of the spleen may cause left-sided hydronephrosis which may become infected and cause the patient to die of sepsis.[1]

Clinical and hematological changes which appear in the subsequent and terminal stages of the disease are unusual polynucleosis, leukemia, thrombocythemia, chlorotic polycythemia, rare anemia, and their combinations. One must not forget that patients can have the disease and not have any subjective symptoms.

It may be generally stated that those patients who have had the disease for a long time and who do not succumb to a vascular complication or later in the erythroleukemic phase, will ultimately reach marked anemia, and in addition there will be leukopenia and thrombopenia. However, the platelets may persist in large numbers, and anemia may then be associated with thrombocythemia.[26] It was found that in 3 cases out of 15, after having polycythemia vera for many years, there was increasing splenomegaly, anemia, and a leukemic blood picture. In one case examined postmortem, the spleen, liver, and bone-marrow showed a hyperplastic blood formation very similar to that seen in myelogenous leukemia.[27]

Minot and Buckman[27] feel that myelogenous leukemia and erythremia appear initimately related, and both may be of a neoplastic nature. There occur cases of erythremia and leukemia that illustrate multiple varieties of varying degrees of primary pathological activity of the myeloid tissues which usually conforms to a definite type. The development of marked anemia in erythremia, as in leukemia, is dependent upon a disorderly proliferation of the myeloid tissue.

Treatment will not be discussed other than to say that since the etiology of the disease is not known, all treatments are symptomatic. The latest and seemingly most effective therapy is with P^{32}.

COMMENT

Polycythemia vera is a rare disease and is often thought to be confined to the blood picture of high red blood cell count, high white cell count, increased hemoglobin, elevated hematocrit, and increased blood volume, with its resultant congestion in the vessels. The patient generally thought of has a red cyanotic appearance, dyspnea on exertion, and splenomegaly.

This picture of the polycythemia vera patient must be expanded to include all of the symptoms, signs, and complications which can be caused by congestion in the lungs, overloading of the heart with resultant chronic passive congestion of the liver, right-sided heart failure, and storage of red blood cells in the spleen. Congestion and increased viscosity of the blood with its retarded flow in the extremities causes peripheral circulatory failure. When one considers the large range of possibilities which may be caused by thrombosis or hemorrhage in the skin, brain, lungs, heart, liver, spleen, kidney capsule, mesenteric vessels, and hemorrhoids, polycythemia vera and its complications must be ruled out in many patients admitted to the hospital.

We must also think in checking a patient that he might have had polycythemia vera for years and that his present condition may be a terminal phase, or that he has been treated for polycythemia vera and the present state may be due to the continued polycythemia vera and to the effect of therapy.

Finally, we must keep in mind that some patients with polycythemia vera live in good health without symptoms. Others have symptoms which cannot be explained by all of the aspects known about polycythemia vera. Lastly, there can be other conditions associated with polycythemia vera that are either not related to the polycythemia vera or are made worse because of the presence of polycythemia vera.

In summary, many of the various symptoms and signs that have been observed in patients suffering with polycythemia vera have been listed.

Some of the more distinctive complications and some illustrative case histories have been given.

The clinical conditions under which the diagnosis

of polycythemia vera has to be ruled out have been stressed.

A number of complications which polycythemia vera patients develop terminally have been presented.

BIBLIOGRAPHY

1. Meakins, J. C.: The Practice of Medicine, Ed. 5, St. Louis, The C. V. Mosby Company, 1950, pp. 551-553.
2. Best, C. H., and Taylor, N. B.: Physiological Basis of Medical Practice, Ed. 5, Baltimore, The Williams & Wilkins Company, 1950, p. 61.
3. Schafer, P. W.: The etiology and treatment of polycythemia rubra vera, Ann. Surg. 122:1098 (Dec.) 1945.
4. Reznikoff, P.; Foot, N. C., and Bethea, J. M.: Etiologic and pathologic factors in polycythemia vera, Am. J. M. Sc., 189:753 (June) 1935.
5. Berlin, H. I.; Lawrence, J. H., and Gartland, J.: Blood volume in polycythemia as determined by P^{32} labeled red blood cells, Am. J. Med. 9:747 (Dec.) 1950.
6. Sturgis, Cyrus C.: Hematology, Springfield, Illinois, Charles C Thomas, 1948, p. 720.
7. Brockbank, T. W.: Neurological aspects of polycythemia vera, Am. J. M. Sc. 178:209 (Aug.) 1929.
8. Downey, Hal.: Handbook of Hematology, New York, P. B. Hoeber, 1938.
9. Brohn, George E., and Griffin, Herbert Z.: Studies of capillaries and blood volume in polycythemia vera, Am. J. M. Sc. 166:489 (Sept.) 1923.
10. Davis, N. S., III: Polycythemia vera (Vaquez-Osler Disease): Gout, angulation of the left ureter with beginning left hydronephrotis, J.A.M.A. 92:1595 (May 11) 1929.
11. Weber, F. P.: Erythraemia with migraine, gout and intracardiac thrombosis, Lancet 2:808 (Oct. 13) 1934.
12. Oppenheimer, B. S.: Vascular occlusion in polycythemia vera, Tr. A. M. Physicians 44:338, 1929.
13. Nagy, F.: Changes in the fundus caused by polycythemia, The British Journal of Ophthalmology 34:380 (June) 1950.
14. Pollack, L. J.: A case of chorea and erythremia, J.A.M.A. 78:724 (Mar. 11) 1922.
15. Lawrence, John, and Rosenthal, Robert L.: Multiple myeloma associated with polycythemia report of four cases, Am. J. M. Sc. 218:149 (Aug.) 1949.
16. Newman, Walter; Feltman, James A., and Devlin, Blanche: Pulmonary function studies in polycythemia vera, American Journal of Medicine 11:706 (Dec.) 1951.
17. Barker, N. W.: Polycythemia vera and chronic pulmonary disease, Arch. Int. Med. 47:94 (Jan.) 1931.
18. Cases from the medical grand rounds from the Massachusetts General Hospital, Case No. 761507, American Practitioner 4:6 (June) 1953.
19. Friedman, G. A.: A hitherto undescribed form of polycythemia vera and its possible relation to duodenal ulcer, chronic pancreatitis, and a disturbance of internal secretions (adrenalin), 84:701 (Oct. 18) 1913.
20.————: The value of polycythemia for the diagnosis of duodenal ulcer, based on 16 operatively demonstrated cases, Med. Rec. 85:875 (May 16) 1914.
21. Wilbur, D. L., and Ochsner, H. C.: The association of polycythemia vera and peptic ulcer, Ann. Int. Med. 8:1667 (June) 1935.
22. Cases from the medical grand rounds from The Massachusetts General Hospital, Case No. 35252. N. Eng. J. Med. 240:1022 (June 23) 1949.
23. Schual, A. R.: Hepatic complication in polycythemia vera, Arch. Int. Med. 62:925 (Dec.) 1938.
24. Cases from the medical grand rounds from The Massachusetts General Hospital, Case No. 791408, American Practitioner 4:5 (May) 1953.
25. Mackenzie, D. W.: Perirenal hematoma primary with polycythemia vera, J. Urol. 23:535 (May) 1930.
26. Rosenthal, N., and Bassen, F. A.: The course of polycythemia vera, Arch. Int. Med. 62:903 (Dec.) 1938.
27. Minot, G. R., and Buckman, T. E.: Leukemia following polycythemia vera, Am. J. Med. Sc. 166:469 (Oct.) 1923.
28. Strobel, Charles F.: Polycythemia vera, Amer. Pract. 4:853 (Dec.) 1953.
29. Sodeman, William A.: Pathologic Physiology: Mechanisms of Disease, Philadelphia, W. B. Saunders Company, 1950, pp. 420-425.

Payment of $76,000 an hour—that is the amount spent by all Blue Cross Plans together for hospital service every hour in 1953. This adds up to $1,800,000 every day, or $331,000,000 for a period of six months. This amount represents 91.7 per cent of the total income of all 86 Blue Cross Plans during that period.

ACTIVITIES OF MEMBERS

Dr. Fred J. McEwen, Wichita, addressed the Kansas State Dental Association at its annual session in May. His subject was "Coronary Heart Disease."

Dr. Paul H. Lorhan, professor of surgery and chairman of the anesthesiology section at the University of Kansas Medical Center, recently received a commemorative plaque and honorary diploma from the International University of Mendez Pelayo. The award was in recognition of his services to the practice of anesthesiology in Spain.

Dr. Clarence H. Benage, Pittsburg, was guest speaker at the May meeting of the Crawford County Medical Assistants' Society. He told of advancements in patient care in the past 105 years.

Dr. Henry G. Hurtig, Hanover, was guest of honor at a breakfast given by the Hanover Council of the Knights of Columbus on May 23. The tribute was in recognition of Dr. Hurtig's 50 years of continuous membership and for his many years of service to humanity as a physician.

Dr. W. C. Goodpasture and Dr. George E. Milbank, Wichita, served on the Budget Hearing Committee of the Wichita Community Chest last month.

Dr. Orville R. Clark, Topeka, addressed the Shawnee County Licensed Practical Nurses' Association at its May meeting on the subject of "Need of Improved Nursing Care Due to Advances Made in Surgery in Recent Years."

Dr. M. D. McComas, Jr., Concordia, presented an exhibit on plastic surgery of the kidney at a meeting of the American Urological Association in New York in May.

Dr. Frances Schiltz, Wichita, was presented a certificate of award by the University of Wichita on May 12 for 25 years of service as medical director for women.

Five Wichita physicians took part in career conferences sponsored by high schools in Wichita before

the close of the school year, each speaking on "Medicine as a Career." The five were: Dr. Harold F. O'Donnell, Dr. Edward S. Brinton, Dr. Condon T. Hagan, Dr. Howard C. Clark, and Dr. Dale W. Peters.

Dr. Lester D. Bowles, Clifton, announced last month the closing of his office in Clifton to move to Norton.

Dr. Martin Hagan, Wichita, recently observed the 50th anniversary of his start in the practice of medicine and was the subject of a feature story in the *Medical Bulletin* of Sedgwick County.

Dr. William J. Smith, who has been practicing in Stockton for five years, recently received orders to report for induction into the armed forces on June 30.

A feature story about Dr. and Mrs. Frank A. Eckdall, Emporia, was carried in the *Emporia Gazette* on May 22 on the occasion of their golden wedding anniversary.

Four Wichita physicians were named to office in the Sedgwick County Chapter of the Kansas Academy of General Practice last month: Dr. George Thorpe, president; Dr. Harold Low, president-elect; Dr. Robert Kasha, vice-president, and Dr. Charles McCoy, secretary-treasurer. Serving on the Board of Directors are Dr. Bruce Meeker, Dr. Jay Wisdom, and Dr. Paul Kaelson.

Dr. Yale E. Parkhurst, Belle Plaine, has received orders to report for active duty with the armed forces in July.

Dr. Edward D. Greenwood, of the Menninger Foundation, Topeka, was leader of a study section on "Our Institutional Treatment Program" at a conference on juvenile delinquency held in Washington this month.

Dr. Carter B. Sigel, Cottonwood Falls, will be called to active duty as a lieutenant in the Navy soon. He plans to return to Cottonwood Falls on completion of a two-year tour of duty.

Dr. H. Preston Palmer and Dr. Galen W. Fields, Scott City, announce that Dr. B. Morris Hopkins will be associated with them in practice after July 7. Dr. Hopkins, a graduate of the University of Kansas School of Medicine, has been interning in Washington, D. C., during the past year.

Dr. John H. Hege, who has been medical director at Sunflower Ordance Works for the past 10 months, moved recently to Hartford, Connecticut, to become medical director for the Royal Typewriter Company.

Dr. Michael J. Cox, Dodge City, addressed the Soroptimist Business Women's Club in Dodge City recently on the subject of "Cancer of the Breast." He also showed a film on self examination.

Dr. Ralph L. Hopp, who formerly practiced in Hardtner, has gone to Ann Arbor, Michigan, for a residency. His practice in Hardtner is being taken over by Dr. Marvin H. Cheshire of Memphis, Tennessee.

Dr. Frederick W. O'Donnell, Junction City, was speaker at a formal ceremony held at Fort Riley in June to dedicate Wainwright Hall in honor of the late General Jonathan Wainwright.

Dr. C. C. Hawke recently returned from Europe where he attended meetings of the Association of Surgeons of Great Britain and Ireland, the American College of Surgeons, and the International Society of Surgeons. He gave an account of his trip when he addressed the Winfield Chamber of Commerce at a meeting held last month.

Dr. Dick B. McKee, Pittsburg, was elected president of the Kansas State Board of Health at its annual meeting held last month. Dr. H. St. Clair O'Donnell, Ellsworth, was named vice-president, and Dr. Thomas R. Hood was reappointed executive secretary.

Booklet on Civil Defense

A booklet recently published by the Council on National Emergency Medical Service of the American Medical Association outlines the part the physician should play in civil defense activities. Reprinted from the *Journal of the A.M.A.*, the articles discuss such aspects as organizing for civil defense, developing medical participation in civil defense, the physician's responsibilities, and the doctor and the improvised hospital.

The booklet may be secured on request.

BLUE SHIELD

Prolonged Illness Coverage by Prepayment Plans

It is now recognized by a large number of people that Blue Cross-Blue Shield plans cannot solve community health problems with programs that limit benefits to services rendered only in the hospital. There is probably little doubt that treatment for minor ailments in the home, doctor's office, or outpatient department of the hospital can be paid for more economically out-of-pocket than they can be insured against. This is not true, however, of the services required by persons suffering from protracted illnesses.

It is not economical from a community point of view to maintain a patient in a hospital bed when the illness or injury no longer requires hospitalization but does probably require follow-up care for a long period of time. Let us not place undue emphasis on services rendered outside the hospital, for it is admitted that the basic services rendered in the hospital are the most important part of any prepayment plan. For this reason, it is feasible to offer prolonged illness coverage only to persons who have basic coverage of both Blue Cross and Blue Shield.

Some nine Blue Shield plans have followed the logical development of experimenting with prolonged illness coverage. It is estimated that these nine plans, along with a few commercial companies, now have close to a million members protected against what is commonly called major medical expense.

Most commercial companies in the field operate on the deductible principle, that is, the member pays the first $100 or $200 and the company pays the balance up to $3,000 or $5,000. Some Blue Shield plans have adopted this principle, while others make a maximum payment for specified conditions with no deductible involved.

ILLNESSES LISTED

Illnesses and injuries representative of those covered by prolonged illness contracts are as follows:

1. Poliomyelitis and its late effects.
2. Cancer, including disseminated cancer such as lymphoma, myeloma, leukemia, and Hodgkin's disease.
3. Benign neoplasms of the brain or spinal cord.
4. Subacute combined degeneration of the spinal cord.
5. Cerebral hemorrhage, embolism, or thrombosis.
6. Coronary embolism or thrombosis.
7. Subarachnoid hemorrhage.
8. Rheumatic fever or chorea.
9. Congestive heart failure.
10. Active pulmonary tuberculosis.
11. Ulcerative colitis and regional enteritis.
12. Cirrhosis of the liver with ascites requiring paracentesis or following a shunt operation.
13. Chronic nephrosis or chronic nephritis with uremia.
14. Pemphigus.
15. Myasthenia.
16. Amputations where prostheses are indicated.
17. Fractures.
18. Hemiplegia, paraplegia, or quadriplegia.

Although this list embraces a wide scope of illnesses, it is hoped that more experience in this field will ultimately lead to coverage for any condition requiring care after discharge from the hospital, even if for a short period.

BENEFITS OFFERED

Each member is entitled to benefits for medical, surgical, hospital, and other related services, varying, of course, with each individual plan's contract. It should be mentioned that plans offering this coverage try to let the member assume some responsibility for items that are likely to be used excessively such as drugs, appliances, special nurses' services, and nursing home care. Physicians and visiting nurses are paid in full.

A. Any member being treated for one of the above listed 18 conditions will receive general benefits as follows: (No attempt has been made in this article to list specific benefits since each plan offers different basic coverage and prolonged illness coverage.)

1. Physicians' Service
   a. For hospital visits after expiration of basic Blue Shield coverage.
   b. For medical (non-surgical) services rendered following discharge from hospital.
   c. X-ray, x-ray therapy, pathology examinations, and physical therapy according to fee schedule of contract in force.
2. Room and Board
   a. Number of available days increased to approximately twice that of basic contract.
   b. About 75 per cent of charges, or a daily indemnity for room in licensed nursing home.
3. Medication, Drugs, Appliances and other Ancillary Services
   a. Usually 75 per cent of medications, drugs, and operating room is covered after expiration of basic coverage benefits.
   b. The same applies to drugs requiring prescriptions after discharge from hospital (75 per cent).
   c. X-ray, x-ray therapy, pathology, and out-patient room according to fee schedule.
   d. The entire cost of rental or 75 per cent of

the cost of purchase of appliances ordered by the attending physician.

4. Nursing Services
   a. About 50 per cent of private duty nursing charges up to a maximum of around $250 to $400.

B. Some plans provide most of the above services to members confined to mental hospitals. In addition, benefits are available for electric shock and insulin shock treatments. This applies, of course, only to patients diagnosed as acute mental rather than as custodial patients.

C. In addition to the 18 categories of conditions listed previously, persons suffering from any other prolonged illness *in the hospital* are given an extension of benefits beyond basic coverage. The amount of additional benefits varies from plan to plan.

Perhaps the success of this type coverage is best assured in those plans where 80 per cent or more of their members are covered by a Blue Shield "Service Benefits" clause. Since, in such cases, the charge made by physicians will be that of the Blue Shield fee schedule, and since the basic contract will cover a great part of many of these cases, the prolonged illness rider can be offered at a reasonable rate and still be self sustaining.

D. Kansas Blue Shield is watching these experiments with interest, and it is expected that tangible steps will be taken soon to introduce discussion of the ideas with Blue Shield Relations Committees.

Last year's traffic toll of deaths and injuries was the heaviest in history.

Number of Physicians High

The total number of physicians licensed to practice in the United States, 218,522, set an all-time high record in 1953. Official figures from the 52nd annual report on medical licensure of the American Medical Association's Council on Medical Education and Hospitals indicate that 7,276 persons were added to the medical profession in 1953. During the same period, 3,412 physician deaths were reported.

The report shows that 14,434 medical licenses were issued in 1953 by the examining boards of the 48 states, the District of Columbia, Alaska, Canal Zone, Guam, Hawaii, and Puerto Rico. Of this number, 6,565 were granted by examination and 7,869 by reciprocity or endorsement.

The present high level of medical education in this country is indicated by the fact that of the 5,646 graduates of approved medical schools in the United States to take examinations, only 3.8 per cent failed to pass. In comparison, however, of the 1,463 graduates of foreign medical faculties examined, 45.5 per cent failed.

Of the total number of physicians in the United States at the close of 1953, 156,333 were engaged in private practice, 6,677 were in research and teaching, 29,161 were interns or residents or physicians engaged in hospital administration, 9,311 were retired or not in practice, and 17,040 were in government service.

JOURNAL readers should patronize JOURNAL advertisers. Without advertising, the JOURNAL could not be published in its present form.

Physician's Obligation—Accident Prevention

Physicians, by including advice on accident prevention in their routine health supervision of the child, will increase the parents' knowledge and understanding of child care and management in general.

When giving accident prevention advice, the physician should keep in mind:

1. Hazards at certain ages.
2. Behavior and drives at certain ages.
3. The emotional attitude of the parents toward the child and toward each other.
4. Physical, emotional and intellectual capacities of the child.
5. The physical and emotional environment of the child.

The private physician has the opportunity on home calls to suggest safety measures and to point out hazards. He also can contribute much to help develop community education by working with safety or health groups such as Parent-Teacher Associations and schools.

There are many individuals who believe that when we place proper emphasis upon accident prevention, giving to it the same amount of study, personnel, funds, etc., as we now give to the prevention of communicable disease, we can expect almost comparable results.

Prepared by Committee on Child Welfare

Clinicopathological Conference

CASE PRESENTATION

Dr. Harold Harvey, resident in obstetrics: This patient was a 59-year-old white living female who was a waitress in a tavern. She was first seen in the medicine outpatient department on January 25, 1954, complaining of "swelling in stomach," malaise, eructation, and flatulence of increasing severity for two months duration. The patient began losing weight eight months prior to admission.

The past history revealed the usual childhood diseases with deafness following measles at age three. A "cyst" had been removed from the right side of the neck in 1935 and, apparently, had recurred and increased in size. She had also had an unexplained anemia while working in the powder room of a soap factory in 1932.

The family history was not remarkable other than that one brother died of carcinoma of the liver.

Review of systems revealed the patient to be edentulous, to have a "sore tongue," and a chronic cough with four to five cups of clear white, foamy sputum per day. Menstrual history revealed menarche at age 16 with regular cyclic periods until menopause at age 46 followed by no further bleeding.

The physical examination revealed a well developed but emaciated white female. Body weight was 84 pounds with height of 60½ inches. Blood pressure was 115/70, pulse 108, temperature 98.6°F. Positive findings were a dark beefy tongue and a small, oval, hard, non-tender nodule on the right side of the neck. Chest examination revealed the right diaphragm to be fixed. Moist, harsh rales were heard in both bases posteriorly. There was dullness to percussion and reduced breath sounds in both bases. Abdominal examination revealed distention. A fluid wave was elicited. Venous congestion was present on the abdominal wall and lower thorax. The abdomen was tympanitic anteriorly but dull in the lateral areas. There was extreme tenderness in the right upper and left lower quadrants with a hard, irregular, fixed tender mass in the left lower quadrant. There was also some tenderness in the left costovertebral angle. The skin was dry, and there was a two-plus pitting edema of the left ankle and pretibial area. Pelvic examination revealed a frozen pelvis.

Laboratory examination showed the hemoglobin to be 11.6 grams; white blood count was 13,000 with a normal differential; VDRL positive twice with 32 Kahn and 32 Kolmer units; NPN 39 mgm. per 100 ml.; creatinine 1.5; sugar 117; sodium 133 mEq./liter; potassium 5.5 mEq./L.; CO_2 22.7 mEq./L.; chlorides 99 mEq./L.; total protein 5.9 Gm./100 ml. with 1.78 Gm. globulin and 4.12 Gm. albumin; sedimentation rate 23 millimeters per hour. EKG and x-rays will be shown.

Hospital course: The patient was admitted to the hospital on February 1, 1954. Paracentesis with removal of 2000 cc. was done on the following day. Pelvic examination after paracentesis revealed the vagina and cervix negative aside from a slight bloody discharge from the cervix. The uterus was not definitely outlined but seemed fixed by a hard nodular tumor mass filling the pelvis.

The patient rapidly became worse and expired at 6:05 a. m. on February 4, 1954, the third hospital day.

Dr. Delp: Any questions of Dr. Harvey?

Q: Were any other blood pressure readings recorded?

Dr. Harvey: The blood pressure reported here is 115/70. That was the admission blood pressure. No other blood pressures were noted in the chart. A paracentesis was done on February 2. The pulse rate was then 140. The tachycardia continued on the 3rd and 4th.

Q: What did the paracentesis fluid look like?

Dr. Harvey: The fluid had a cloudy yellowish appearance.

Q: Was the fluid examined for cells?

Dr. Harvey: It was sent to the pathology laboratory.

Dr. Delp: The report read, "A large number of type 5 cells." Any other questions?

Q: Was there any tenderness in the left leg?

Dr. Harvey: No tenderness in the leg was mentioned.

Q: How long had the patient had the chronic cough and sputum?

Dr. Harvey: Her main symptoms had started two months before and fairly rapidly became worse. However, she had worked until December.

Dr. Delp: There is no specific reference either in the history taken in the clinic or on three histories in the hospital as to how long the patient had had this cough. I gathered from other notes that she had had the cough about two months.

Q: What was her actual red blood count?

Dr. Delp: 4,380,000.

Q: Can you elaborate on the abdominal symptoms?

Dr. Delp: After eating a very small amount, the patient felt diffuse abdominal discomfort. She couldn't exactly localize it. She had the discomfort even with taking water.

From the University of Kansas Medical Center. Edited by Glen R. Shepherd, M.D., and Mahlon Delp, M.D., from recordings of the conference participated in by the departments of medicine, obstetrics, radiology, pathology, and the third and fourth year classes of medical students.

Q: What were her bowel habits?

Dr. Harvey: No change. She had always been somewhat constipated. However, she did state that in recent weeks it felt as though she was having difficulty with her bowel movements.

Dr. Max Allen, associate professor of medicine: Can we assume that the parts of the physical examination not mentioned were negative?

Dr. Delp: Yes. This patient had a high degree of deafness, and it was rather difficult to communicate with her.

Q: What were the urinary findings?

Dr. Delp: Specific gravity 1.025, acid reaction with heavy trace of albumin, negative sugar, 10 to 20 pus cells per high power field. After paracentesis, a note on the chart states that the abdomen was somewhat softer and that the liver, spleen, and kidneys were not palpable.

Q: Could you elaborate on the patient after she became worse in the hospital?

Dr. Harvey: The pulse rate, as I mentioned, went up to rapid levels, the respirations increased, and the temperature gradually went down to subnormal levels. Dr. Hunter mentioned that he felt she was semicomatose.

Q: Was her blood pressure taken when she had a pulse of 140?

Dr. Harvey: No.

Q: Was she jaundiced?

Dr. Delp: No, nor had she been jaundiced at any time in her past.

Q: Was there any hemoptysis during her hospital course?

Dr. Harvey: No.

Q: Was there any chest pain?

Dr. Delp: No chest pain. She was quite dyspneic before death but had no chest pain. She was specifically questioned about it and denied any chest pain. This patient was complaining only of gastrointestinal symptoms. Any other questions? We have an EKG and x-ray films. Dr. Dimond, do you have any comments about the EKG?

Dr. E. Grey Dimond, professor of medicine: I think it is a normal EKG other than the sinus tachycardia. The Q waves are probably normal Q waves. The T wave is a bit broad. I read this as sinus tachycardia.

Dr. Delp: All right, thank you. Mr. Nystrom.

Mr. Curtis Nystrom, fourth year student:* We have a chest x-ray which was taken on the second day of hospitalization. The lungs are not congested in the apices. The angles are clear. There seems to be quite a bit of infiltration in both lower lobes with even more infiltration on the right side, which may be a little bit of bronchiectasis. The diaphragm is elevated, probably by fluid in the abdomen. The aorta is not widened. There does not seem to be any bone pathology. We don't see air under the diaphragm. The left lateral film doesn't add a whole lot. It does show some patchy infiltration in the lower part of the lung. The heart isn't enlarged.

Flat plate of the abdomen shows a haziness from the fluid. The bowel loops seem higher than usual as if they were displaced from the pelvis. I wouldn't definitely say what was holding the bowel out of the pelvis. It could be either some mass in the colon or a uterine or even an ovarian mass.

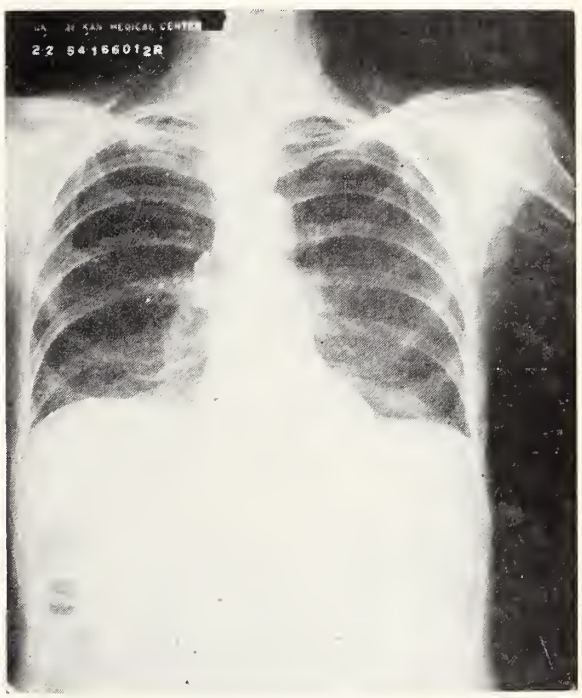


Figure 1. Chest x-ray showing elevated diaphragm and patchy atelectasis of lower lobes.

Dr. Delp: Dr. Tice, do you have any comments concerning this interpretation?

Dr. Galen Tice, professor of radiology: Dr. Litton's impression was that the changes in the chest were likely due to the high diaphragm with segmental or patchy atelectasis. I think the interpretation of the the abdomen is entirely correct.

Dr. Delp: We have a 59-year-old patient who presented herself to the outpatient department. We assume this patient walked in, gave her history, was examined, was referred to another clinic within the outpatient department. Then only a matter of a few days later she re-entered the hospital and within three days was dead.

The patient complained chiefly of gastrointestinal symptoms, discomfort, probably cramp-like pain, throughout the abdomen, precipitated by eating or drinking. She had some loss of appetite. She was able to eat only small amounts of food at a given

* Received M.D. degree, June 7, 1954.

time, because eating precipitated discomfort. A discomfort of distention in the rectum annoyed the patient considerably. She had a cough and had been bringing up a large amount of sputum—we're not certain for how long but at least for a couple of months. Her weight loss may be a little unreliable as far as the patient's recorded history is concerned.

At the time she entered the outpatient department her weight was recorded as 84 pounds. This patient had a lump in the neck removed in 1935. She said she now had a recurrence of a lump in her neck. Whether or not it was actually recurrent, I am not sure. It might be an entirely new lesion. After she entered the hospital, she had a paracentesis. So called type V cells were detected in this fluid. We



Figure 2. Flat x-ray film of the abdomen showing haziness due to fluid and displacement of bowel loops from pelvis.

need to explain what happened in such a relatively short period of time. Spearman, may we have your differential diagnosis?

DIFFERENTIAL DIAGNOSIS

Mr. Jesse Spearman, fourth year student:* We have here today the case of a 59-year-old white female with the chief complaints of flatulence, eructation, and distention of the abdomen for two months duration and a weight loss for eight months. As Dr. Delp said, her weight at the time she came in was 84 pounds. That is pretty small. I could assume that she probably was small from the start of her symptoms. She also had a mass in the abdomen with unilateral edema of the leg. She was not complaining of pain. She had 32 Kolmer units and a positive VDRL. She had a distended abdomen, characteristic of ascites. I wish to base my differential diagnosis on the causes of ascites with a mass in the pelvis.

First, I'd like to consider the cardiac diseases. According to Cabot, 50 per cent of all ascites is caused by cardiac diseases. In this case we had no symptoms or physical findings that would lead me to believe that she had a cardiac lesion. I think on the basis of the history and physical findings, we can rule out cardiac disease as the cause of this patient's ascites.

The next I'd like to consider is glomerulonephritis. This is a common cause of ascites, but usually in glomerulonephritis individuals have presenting symptoms of edema elsewhere before complaining of ascites.

Liver disease is a common cause of ascites. You have to consider cirrhosis. This individual probably didn't eat very well. She could also have had a specific infection of the liver by syphilis.

Tuberculous peritonitis is another common cause of ascites. Tuberculous peritonitis in children is the commonest cause of ascites, but here x-ray examination was negative for pulmonary tuberculosis. In order to diagnose tuberculous peritonitis, I'd expect to have findings in the x-rays positive for tuberculosis.

Another consideration is neoplastic peritonitis. In patients of middle life or past, metastasis to the peritoneum is a common cause of peritonitis.

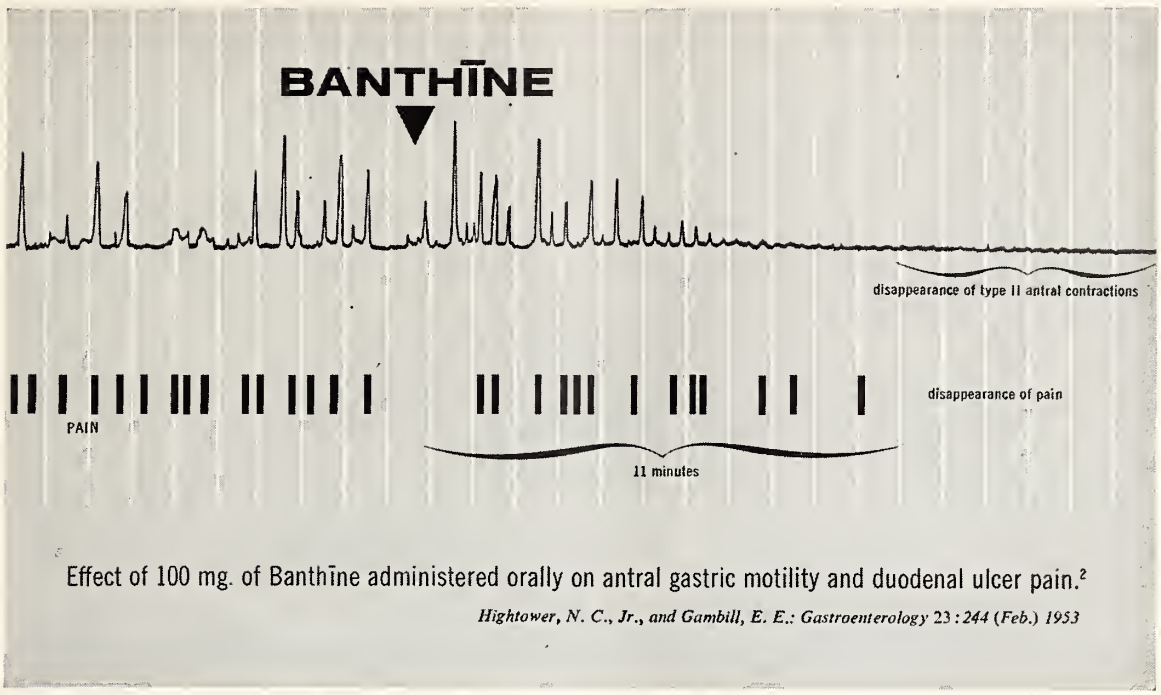
Nutritional edema could cause ascites. I think, if this were a nutritional edema, the serum protein reports in this case would be different. The total protein was 5.9 with a good albumin-globulin ratio.

Hemoperitoneum might be considered. One of the most frequent causes of hemoperitoneum is a rupture of ectopic pregnancy. This patient was 59-years-old and past the menopause, so I don't think she could have an ectopic pregnancy.

Other considerations are severe anemia, pernicious anemia, aplastic anemia, leukemia, or Hodgkin's disease. The laboratory and physical findings in this patient do not suggest these diseases.

Last I'd like to consider ovarian neoplasm. Ovarian cysts will give dullness in the midabdomen. Here tympany of the midabdomen was found. Ovarian fibroma is one of the rarities, but if you never think of it you'll never make the diagnosis. Very frequently ascites occurs and even hydrothorax. Another ovarian neoplasm I'd like to consider is a metastatic neoplasm referred to as Krukenberg's tumor. Krukenberg's tumor can have an origin in any place in the gastrointestinal tract: in the stomach, large intestine, small intestine, or even the gallbladder. If I were to consider Krukenberg's tumor seriously, I'd think mostly of a metastasis from the gallbladder, but in this patient there was no history of any gallbladder disease.

* Received M.D. degree, June 7, 1954.



Effect of 100 mg. of Banthine administered orally on antral gastric motility and duodenal ulcer pain.[2]

Hightower, N. C., Jr., and Gambill, E. E.: Gastroenterology 23:244 (Feb.) 1953

Banthine® Reduces Hypermotility and Hyperacidity in Peptic Ulcer

A recent evaluation of anticholinergic therapy in peptic ulcer emphasizes the fact that now the profession has at its disposal agents that are "effective in reducing both secretory and motor activity of the stomach."

The effect on motor activity is generally more pronounced and less variable than on secretion; pain relief is usually prompt; a high degree of effectiveness is noted in ambulatory ulcer patients.

Ruffin, J. M.; Texter, E. C., Jr.; Carter, D. D., and Baylin, G. J.: J.A.M.A. 153:1159 (Nov. 28) 1953.

With its proved anticholinergic effectiveness, Banthine has been found extremely useful in the medical management of active peptic ulcer, whether duodenal, gastric or marginal.

The immediate increase in subjective well-being and the simplicity of the Banthine regimen assures patient cooperation. The recommended initial therapeutic dose is 50 or 100 mg. (one or two tablets) every six hours around the clock, with subsequent individual adjustment. The usual measures of diet regulation, rest and relaxation should be followed.

Banthine is effective in other conditions caused by excess parasympathetic stimulation. These include hypertrophic gastritis, acute and chronic pancreatitis, biliary dyskinesia and hyperhidrosis. Banthine is contraindicated in the presence of glaucoma and should be used with caution in the presence of severe cardiac disease or prostatic hypertrophy.

Banthine® bromide (brand of methantheline bromide) is supplied in scored tablets of 50 mg. and in ampuls of 50 mg. It is accepted by the Council on Pharmacy and Chemistry of the American Medical Association. G. D. Searle & Co., Research in the Service of Medicine.

Carcinoma of the ovary includes the functional neoplasms. Ten per cent of all the solid neoplasms of the ovary are granulosa cell or theca cell tumors or luteomas. These particular neoplasms appear earlier in life, usually before the menopause. There have been cases reported after the menopause, but they are rare. There are other neoplasms such as pseudomucin or serous cyst adenoma. I don't think the physical findings of a hard nodular mass fit these.

In conclusion, I think that the primary diagnosis is secondary ovarian neoplasm from some place in the gastrointestinal tract, probably the sigmoid colon. Neoplasm of the sigmoid colon will give unilateral edema of the legs. There are many other questions that probably need answering. The stasis or the immobility of the diaphragm on the right side is one. Regarding the distention of the abdominal veins, I think this particular patient could very well have had more than one disease. With carcinoma of the sigmoid there could have been thrombosis of the portal system which would produce back pressure and the distention of the abdominal veins. Likewise, the hemorrhoids that have been reported could occur.

Dr. Delp: Thank you, Spearman.

Dr. Hunter, associate in gynecology and obstetrics: A fair amount of the patient's light weight was made up of fluid in the abdomen. Anyone who has a mass in the pelvis with ascites and a history like this patient gives probably has a pelvic malignancy. By that I mean ovarian malignancy. It might be noted that the main part of the nodular mass in the pelvis was on the left side. We interpreted that to be massive enough to impair circulation returning from the left leg, and we accounted for edema on that basis. Regarding the possibility of Krukenberg's tumor that has been mentioned, it's stated that Krukenberg's tumors are usually bilateral. They tend to retain the shape of the ovary, and that was not present in this case. No definite ovarian mass as such could be felt in this patient, even after the paracentesis, so I think that that would be against Krukenberg tumor. I think that the gastrointestinal complaints she had probably were secondary to ascites and were not primary. As for the beefy red tongue, she gave a poor dietary story, I think. It was difficult to talk to this patient because she was hard of hearing and very sick.

Our diagnosis at the time was ovarian malignancy with carcinomatosis, ascites, and probable pleural implant or pulmonary metastasis in spite of the negative chest x-ray. I think the terminal event was bronchopneumonia. This patient's terminal course is not very well documented, as you readily see. From what we know about it, the patient had physical signs of some pulmonary pathology on admission, and her course in the hospital was a steadily worsening one. She did tend to vomit near the end. I think that probably aggravated it, and I would be much in favor of bronchopneumonia as the terminal event causing her death.

Dr. Delp: Dr. Wheeler, you did make a diagnosis in the outpatient department of left ovarian malignancy. Would you document that for us?

Dr. John H. Wheeler, assistant clinical professor of medicine: The diagnosis, of course, was made on the fact that she was obviously chronically ill and she had an easily palpable mass in the abdomen and ascites. Pathologically, we could assume that she had peritoneal implants, and we came to that conclusion because of the absence of other findings. She was sent to gynecology clinic that same day.

Dr. Delp: I'm just never satisfied with this diagnosis of malignancy as being the cause of death. I know I belabor this until it must annoy some of you, but there must be another explanation as to why this patient died. I've seen patients live with intra-abdominal malignancies for two and three years. There must be an explanation for this patient's sudden death. Dr. Allen, do you have any ideas?

Dr. Max Allen, associate professor of medicine: Well, I think I'll go along with the student. I think unilateral edema of the leg with no known explanation for obstructive venous insufficiency of that leg would be a good reason to presume venous thrombosis and subsequent pulmonary embolus. I would like also to comment that the cough and excessive sputum as here described, even with negative chest films, can occur with diffuse lymphogenous metastasis to the lung, with carcinoma of the stomach being perhaps the most common primary site. I think the terminal event must have been a pulmonary embolus. I realize that withdrawal of ascitic fluid with depletion of body fluid and electrolytes can be the reason for a low sodium syndrome developing, producing death by the depletion of circulating blood volume. I would think there would be some other indication of that before the sudden death of the patient.

PATHOLOGY REPORT

Dr. Hans Peters, resident in pathology: The patient appeared remarkably older than her given age. Though we saw the little nodule on the neck, it was not our impression that it was anywhere near the phrenic nerve. It was much higher up, and unfortunately we were not able to take a section of it. We had the impression it was a fibroma.

The abdomen of this patient was markedly distended, and when it was opened we were able to obtain 3500 cc. of serosanguineous fluid. Some of the loops of the bowel were discolored, black bluish red, and there was fibrinous exudate over the entire mesentery and peritoneum.

First of all, I'd comment on the mass. This was found in the left lower quadrant and in the left

Thank you doctor for telling mother about...





The Best Tasting Aspirin you can prescribe

The Flavor Remains Stable down to the last tablet

15¢ Bottle of 24 tablets (2½ grs. each)

We will be pleased to send samples on request

THE BAYER COMPANY DIVISION of Sterling Drug Inc., 1450 Broadway, New York 18, N. Y.

pelvis. It was about 13.5 cm. by 12 cm. by 6.5 cm. It could very well have compressed the left iliac vein. It was connected to the uterus by direct extension by the tube, which split the tumor into two halves. The lower part was placed in the pelvis, and

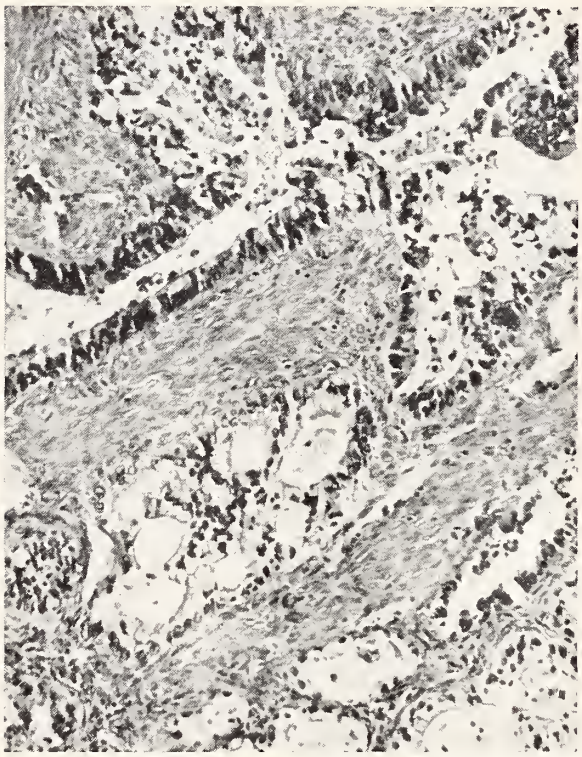


Figure 3. Hematoxylin and eosin stained section of adenocarcinoma of the left ovary showing mucus-producing glands, particularly in the lower portion.

the upper part was above the tube and more in the lower quadrant. The uterus itself was not markedly enlarged. There appeared to be some necrosis of the endometrium of the right tube and of the right ovary.

To look for a cause of the discoloration of the bowel, careful dissection of the superior mesenteric artery was done. A thrombus formation was found in branches of the superior mesenteric artery with infarction of two-thirds of the bowel, as far as to the ileum. The aorta showed marked arteriosclerotic changes with a few ulcerations. On one of the ulcerations, which was just about the iliac axis, thrombus formation was noted. This might very well have been the cause for thrombosis of the superior mesenteric artery. There was infarction of the left kidney and infarction of the spleen. Both of them were small.

The findings in the lungs were restricted to the slides, probably a pneumonic process.

Dr. Harlan I. Firminger, associate professor of pathology and oncology: I have only a little to add from the histological examination to what Dr. Peters has already told you. Sections of the ovarian tumor all showed a glandular type of carcinoma, with tall columnar cells and some pseudostratification, and in some areas there were actually papillary-like projections.

In other areas the adenocarcinoma cells had clear cytoplasm with basally located deeply staining nuclei, and the cytoplasm appeared to be filled with a mucinous-like material. Typical signet cells were evident in many areas.

A PAS stain showed the mucin content of these cells in the tumor. Actually, mucinous material was also evident grossly.

Section of the myometrium revealed small glandular structures infiltrating the myometrium throughout its entire depth, including invasion of venules. There were no solid masses of tumor anywhere in the uterus. The endometrium was thoroughly necrotic over a wide area for some peculiar reason, and there was apparent thrombosis of vessels. This appeared to be infarction of the endometrium of a rather diffuse nature.

The liver showed some periportal fatty change, pretty much restricted to the portal areas.

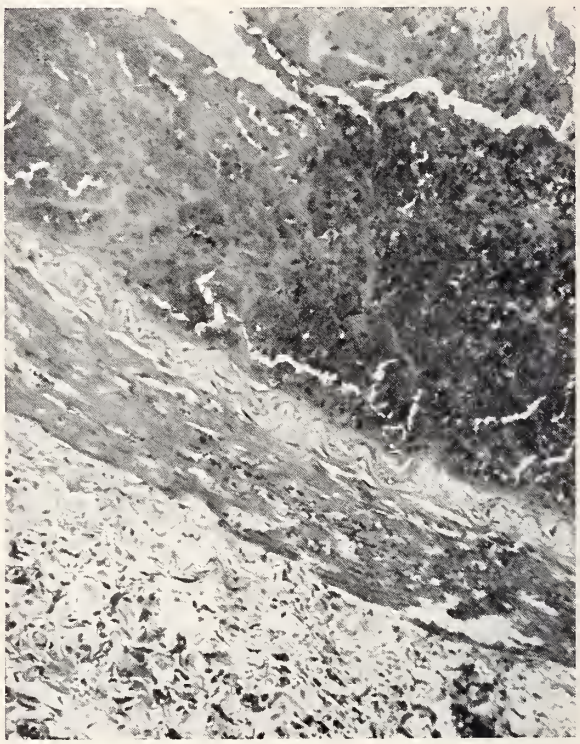
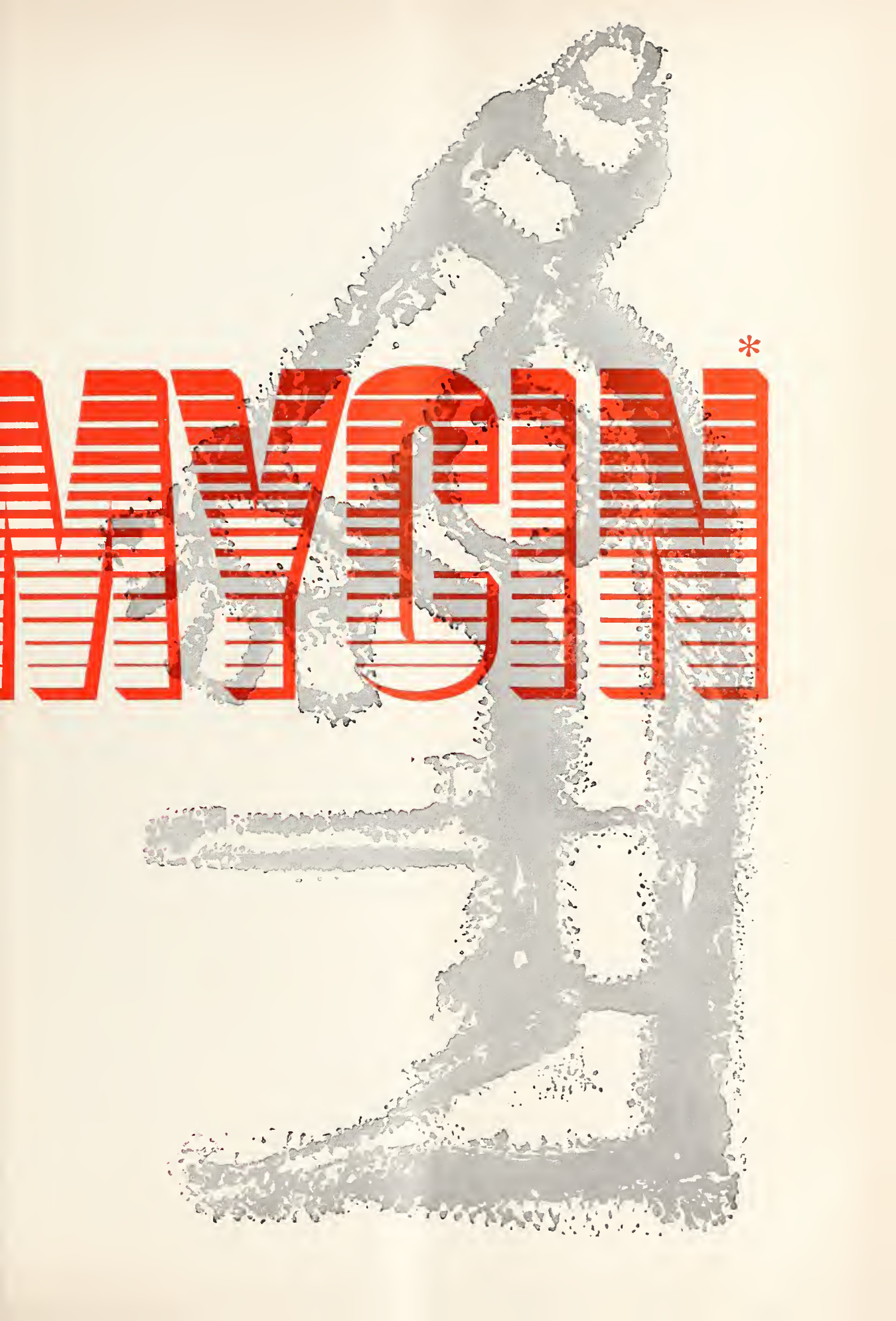


Figure 4. Hematoxylin and eosin stained section of mesenteric artery containing recent embolus showing beginning polymorphonuclear infiltration in the muscular coat of the artery.

Section of the aorta in the area of ulceration above the origin of the celiac axis revealed ulceration of a tremendous atherosclerotic plaque.

The kidneys showed recent infarcts.

The superior mesenteric artery contained recent


MMM*

thrombotic material. Multiple sections revealed what looked like a little fibroblastic content in some areas. A few fibroblasts were suggested, but they were questionable microscopically. That the thrombosis was not post mortem was quite evident because of the polymorphonuclear infiltration in the media of the vessel.

In summary, this is a patient who presented herself with a primary ovarian carcinoma of the left ovary which had not metastasized widely. We found no evidence of metastases with the exception of the extension in lymphatics and interstitially in the uterus immediately adjacent to the tumor. Actually one would consider in the differential diagnosis the possibility that this arose in the uterus. However, its pattern in the uterus was very much against this, being just individual little glands here and there and in vascular structures. There was no isolated mass in the endometrium. Actually, if some one had told me this endometrium had been irradiated I would have believed it because it appeared like the necrotic picture of irradiated endometrium and thrombosis of vessels.

But knowing this was not done, it seemed that that must have been due to vascular obstruction and an unusual infarction of the endometrium, at least in some areas. Then, as Dr. Delp tried his best to point out, something happened to this patient in addition to malignancy, something entirely unrelated to the presence of malignancy unless emaciation and debility are important.

What role paracentesis and examinations may have had, I can't say. She did have arteriosclerosis of the aorta with ulceration and plaque formation. Within a few days, I would suggest four to seven days, before her death, I presume that a portion of the mural thrombus in the aorta broke loose and into several pieces. This may have occurred more than once because the lesions in the spleen and kidneys appeared slightly older. The lesion in the bowel, I would guess, happened two or three days ante mortem. These bits of mural thrombus broke loose and lodged in the arteries with orifices just below this area. This produced infarction. Subsequent symptoms were primarily due to the lesion in her bowel from acute infarction of the bowel. Shock developed. I'm sorry blood pressures weren't taken, but I'm sure she must have had shock level blood pressures. The pulse rates would bear that out. In addition, a certain amount of peritonitis occurred. How much it was related to paracentesis and how much was secondary to leakage through this degenerating bowel, I cannot say. I think it could be secondary to the infarction.

Dr. Delp: I'm completely unabashed in continuously championing the idea of studying all patients really carefully, sincerely, and conscientiously from a clinical standpoint. I don't mean that you have to subject such patients as this to all sorts of gadgeteering to examine them, but I think you should do a meticulous physical examination. I think you should be thoughtful about the findings, and I'm particularly talking about patients with malignant disease. It is hoped that some time in the future we will be handed some sort of substance or some sort of method whereby we can cure malignant disease. I think a good many clinicians are really going to be in a bad way with the fatalistic attitude. The presence of neoplastic disease is not a satisfactory nor complete explanation for death in many cases—herein is such a case.

ANATOMICAL DIAGNOSIS

Primary

Arteriosclerosis of the cerebral and coronary arteries, slight, with one area of almost complete occlusion in the anterior descending branch of the left coronary artery; arteriosclerosis of the aorta, advanced, with ulceration of the abdominal aorta 2 cm. superior to the origin of the celiac axis.

Recent embolic occlusions of the superior mesenteric artery and a branch of the splenic artery.

Recent hemorrhagic infarction of the jejunum and ileum and small hemorrhagic infarcts in the left kidney and spleen.

Acute dilatation of the stomach, duodenum, and first part of the jejunum with partially digested food and brownish-green material, estimated 2000 ml. volume.

Multiple acute erosions of the mucosa of the stomach.

Acute fibrinous peritonitis with localized abscess formation adjacent to the descending colon 10 cm. below the splenic flexure.

Serosanguineous fluid in the peritoneal cavity, 3,500 ml.

Bronchopneumonia of the lower lobes of both lungs, moderate.

Gastric contents and mucus in the treachea and in both main bronchi.

Recent unhealed puncture wound in the lower anterior abdominal wall (history of paracentesis two days before death).

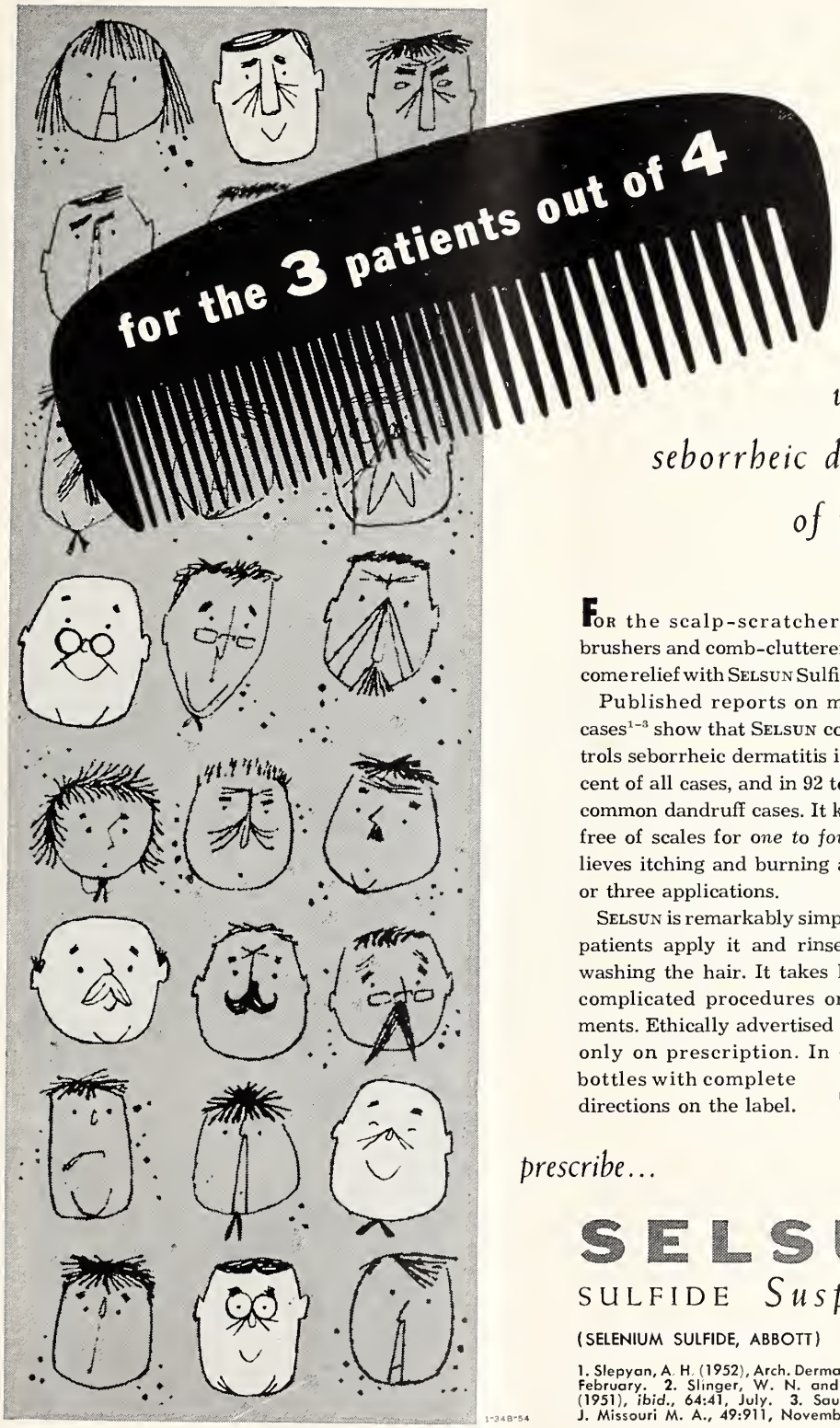
Recent unhealed puncture wounds in the antecubital fossae (history of venipuncture three days before death).

Adenocarcinoma of the left ovary with fibrous adhesions to the left tube, pelvic wall, and to part of the ileum, and extension of tumor into the uterus and invading small vessels in the myometrium.

Thrombosis of veins in the myometrium with necrosis of endometrium.

Submucous leiomyomas in the wall of the vagina and in the uterus.

Hypertrophy and dilatation of the urinary bladder



who have seborrheic dermatitis of the scalp

FOR the scalp-scratchers, shoulder-brushers and comb-clutterers, there's welcome relief with SELSUN Sulfide Suspension.

Published reports on more than 400 cases[1-3] show that SELSUN completely controls seborrheic dermatitis in 81 to 87 percent of all cases, and in 92 to 95 percent of common dandruff cases. It keeps the scalp free of scales for *one to four weeks*—relieves itching and burning after only two or three applications.

SELSUN is remarkably simple to use. Your patients apply it and rinse it out while washing the hair. It takes little time. No complicated procedures or messy ointments. Ethically advertised and dispensed only on prescription. In 4-fluidounce bottles with complete directions on the label. Abbott

prescribe...

SELSUN®

SULFIDE *Suspension*

(SELENIUM SULFIDE, ABBOTT)

1. Slepyan, A. H. (1952), Arch. Dermat. & Syph., 65:228, February. 2. Slinger, W. N. and Hubbard, D. M. (1951), *ibid.*, 64:41, July. 3. Sauer, G. C. (1952), J. Missouri M. A., 49:911, November.

1-348-54

with trabeculation, slight (history of nocturia for unspecified period).

Emaciation (history of 15 pounds weight loss over a period of eight months).

Brown atrophy of the heart, moderate, weight 230 gm.

Accessory

Foci of fibrosis in the myocardium.

Bullous emphysema of the middle and upper lobe of the right lung and the upper lobe of the left lung, moderate.

Fibrous pleural adhesions at the apex of both lungs and in the posterior region of the lower lobe of the right lung.

Calcified lymph nodes at the hilus of the right lung, substernally, near the right first interspace, and beside the aorta near the bifurcation of the trachea.

Cortical adenoma of right adrenal, small.

Retention cyst of the lower pole of the left kidney.

Focal area of fibrosis in the petrous portion of the temporal bone (history of measles followed by deafness 56 years before death).

Subcutaneous nodule in the right lateral region of the neck.

Atrophy of both breasts, moderate.

Hemorrhagic areas over both patellae.

Edentia.

COUNTY SOCIETIES

At a meeting of the Sedgwick County Society held on May 11 the following officers for 1955 were elected: president, Dr. George E. Cowles; vice-president, Dr. Ernest W. Crow; secretary, Dr. Charles P. McCoy; treasurer, Dr. Don E. Miller; censor, Dr. Lennel I. Wright; three-year terms on the board of directors, Dr. Leo K. Crumpacker, Dr. Harold F. O'Donnell, and Dr. Charles L. Woodhouse.

A joint meeting of the Bourbon County Society and the Mercy Hospital staff of Fort Scott was held at Fort Scott in May. Dr. Rollin R. Nevitt reported on the annual meeting of The Kansas Medical Society.

Dr. Charles Hunter, associate in obstetrics and gynecology at the University of Kansas Medical Center, was guest speaker at a meeting of the Labette County Society held at Parsons in May. He discussed new mechanical devices used in his specialty.

THE KANSAS PRESS LOOKS AT MEDICINE

Editor's Note. In this section the JOURNAL *reproduces editorials relating to medicine which have appeared in the lay press. An effort is made to include both favorable and unfavorable comments, and the Editorial Board in no instance assumes responsibility for the opinions expressed.*

The A.M.A.

Some people seem to have the idea that the American Medical Association, as the representative of the medical fraternity in this country, does little or nothing except fight socialized medicine—that, in short, it takes a negative, not a positive, attitude towards medical problems.

The Louisville, Ky., *Courier-Journal* decided to find out just what does go on in A.M.A. headquarters. It sent a staff writer, Robert Clark, to investigate. And it featured a long article by him which contains facts of interest to us all.

Some 60 per cent of all the association's revenues are spent for scientific work. It prints nine medical journals, scores of books, and thousands of pamphlets, all devoted to the war against disease and related matters. It conducts extensive laboratory tests in the fields of chemistry, microbiology and physical medicine. It analyzes widely used products, ranging from foods to cosmetics. It rates hearing aids and other devices for acceptability. It maintains files on illegal practitioners, quacks, and fake and dangerous medicines. One of its newest activities, begun in 1952, has to do with the ever-increasing problem of mental health. A program of study in nine mental health fields is going on. It sponsors a foundation which has given close to $5,000,000 to the nation's medical schools since 1951. It maintains medicine's largest reference library.

So it goes, in Clark's vivid and factual article. And at the end he quotes this significant paragraph from a statement made by an A.M.A. official: "Our most urgent effort should now be directed to the solution of the problem of the medically indigent and the chronically ill. We believe that this objective can be reached without major change in our existing mechanism."—*Atchison Globe, May 27, 1954.*

Family Doctor Still on the Job

The claim has often been made that the practice of medicine has developed into specialization. That

for greater safety in streptomycin therapy...

DISTRYCIN

Squibb Streptoduocin
Streptomycin and dihydrostreptomycin in equal parts

Distrycin has an important advantage over streptomycin. It has the same therapeutic effect but ototoxicity is greatly delayed. Since the patient is given only half as much of each form of streptomycin as he would have on a comparable regimen of either one prescribed separately, the danger of vestibular damage (from streptomycin) or cochlear damage (from dihydrostreptomycin) is significantly lessened.

Signs of vestibular damage appear in cats treated with Distrycin as much as 100 per cent later than in animals given the same amount of streptomycin.

Cat treated with streptomycin shows no nystagmus after whirling.



Cat given the same amount of Distrycin has normal reflex.



On dosage of 1 Gm. per day for 120 days, ototoxicity was as follows*:

	Vestibular damage % of patients		
	Mild	*Moderate*	*Total*
Streptomycin	12	6	18
Dihydrestreptomycin	6	0	6
Distrycin	0	0	0
	Cochlear damage % of patients		
	Mild	*Moderate*	*Total*
Streptomycin	0	0	0
Dihydrostreptomycin	12	3	15
Distrycin	0	0	0

*Heck, W.E.; Lynch, W.J., and Graves, H.L.: Acta oto-laryng. 43:416, 1953.

Distrycin dosage is the same as for streptomycin. In tuberculosis the routine dose is 1 Gm. twice weekly, in conjunction with daily para-aminosalicylic acid or Nydrazid (isoniazid). In the more serious forms of tuberculosis, Distrycin may be given daily, at least until the infection has been brought under control.

SQUIBB

a leader in streptomycin research and manufacture

'Distrycin'® and 'Nydrazid'® are Squibb trademarks

Distrycin is supplied in 1 and 5 Gm. vials, expressed as base

the family doctor had disappeared, much to the detriment and discomfort of the average family. The family doctor has always been in the front line of defense against illness, and has won for himself a secure place in American hearts. Always ready and willing to answer calls day or night, he was more concerned with doing good than in making money.

In addition to having a broad knowledge of all of the physical aspects of disease the family doctor was usually a counselor, confidant and friend. His services went far beyond the practice of his profession, and he became a ready help in time of trouble, sickness and otherwise.

Now it seems the family doctor is still on the job, despite these rumors. Some 4,000 of them, gathering from all parts of the nation, met in Cleveland recently under the auspices of the American Academy of General Practice. For three days those in attendance discussed every phase of a general practice necessary to meet the medical, emotional and spiritual requirements of his patients. He must keep abreast of new discoveries in the field of pathology, that those in need of special treatment can be advised in time.

It was brought out at the meeting that the physical health of the American people is better than their emotional health, as indicated by the fact that 18 tons of aspirin are consumed every day in the United States. The family doctor is still on the job, and the nation will be all the better for it.—*Wichita Eagle, April 3, 1954.*

BOOK REVIEWS

Mayo Clinic Diet Manual, Second Edition. By Committee on Dietetics, Mayo Clinic. Published by W. B. Saunders Company. 247 pages. $5.50.

In reviewing this manual we have found it to be somewhat disappointing. The material is presented in what is generally considered to be an outmoded method. That is, the diets are classified according to the specific condition for which they are to be used. This seems to imply that for each condition discussed these are the only diets which can be used and that these diets cannot be used in other conditions unless repeated in those sections. This type of presentation is misleading to those who use the manual as reference in that not all the diets which may be used can be found in the proper section (e.g., high or low protein levels are found only in the section on children's diets). Also, not all conditions for which dietary treatment may be necessary can possibly be listed.

This manual includes some materials which we would consider to be superfluous, such as the use of a table of caloric values.

It is also a little surprising to find that the dietary regime for diabetic patients advocated by the American Diabetes Association and the American Dietetic Association has not been adopted by the Mayo Clinic, though this is not of necessity the fault of the authors of this manual.

The information given on each specific diet is complete, with the approximate composition of each diet given along with a discussion of the adequacy of the diet. The types of foods to be included and excluded are given, as well as the dietary pattern and sample menu. We do feel, however, that there would be a distinct advantage in having the dietary pattern given first, followed by the foods to be included and excluded. Otherwise, the outlines for each diet appear adequate and clearly outlined.—*E.M. and A.P.*

A Doctor Talks to Women. By Samuel Raynor Meaker. Published by Simon and Schuster, 630 Fifth Avenue, New York. 231 pages. Price $3.95.

Dr. Meaker has written a comprehensive, readable book on the functioning of the female body. This book could be a useful reference in college-level classes in hygiene, marriage-counseling, etc., and should be readily understood by the average intelligent adult woman. It includes definitions of medical terms.

Chapters 9, 10, and 11, titled respectively, "Conception and The Beginning of Pregnancy," "Fertility and Sterility," and "Help for Childless Couples" are especially rewarding and should lead sterile couples to a better understanding of their plight and what may be done to remedy it. The chapters on cancer, tumors and operations will also have a reassuring effect upon the reader.—*C.B.*

Hospital Service in U. S.

Hospitals set a new record during 1953 with 19,869,061 patient admissions as compared with 18,914,847 in 1952, according to the 33rd annual report on hospital services prepared by the A.M.A.'s Council on Medical Education and Hospitals.

For the second time in history, the report states, the number of hospital births exceeded the three million mark, 3,307,182, representing approximately 84 per cent of the estimated 3,910,000 annual births in the United States.

The A.M.A. registered 6,840 hospitals, 4,704 as non-governmental. Gains in patient admissions were noted in both groups but were more pronounced in the non-governmental division credited with 74 per cent of all admissions.

Bed capacity in registered hospitals increased by

50 *and seven*

YEARS TREATING ALCOHOL AND DRUG ADDICTION

In 1897 Doctor B. B. Ralph developed methods of treating alcohol and narcotic addiction that, by the standards of the time, were conspicuous for success.

Twenty-five years ago experience had bettered the methods. Today with the advantages of collateral medicine, treatment is markedly further improved.

The Ralph Sanitarium provides personalized care in a quiet, homelike atmosphere. Dietetics, hydrotherapy and massage speed physical and emotional re-education. Cooperation with referring physicians. Write or phone.

The

RALPH

SANITARIUM

A Unit of the Benjamin Burroughs Ralph Foundation for Medical Research

Telephone VIctor 3624



529 HIGHLAND AVENUE • KANSAS CITY 6, MISSOURI

Ralph Emerson Duncan, M.D., Medical Director.

31,399 over the previous year—1,113,004 in governmental and 460,010 in non-governmental hospitals. Average length of stay per patient was reduced in general hospitals from 9.8 to 9.3 days. Federal general hospitals also showed a reduction—from 26.2 to 23.6 days.

Eight hundred fifty hospitals are approved for internships, 1,146 for residencies, and 1,071 for professional nursing education.

ANNOUNCEMENTS

The International Academy of Proctology announces its annual cash prize and certificate of merit award contest for 1954-1955. An award of $100 will be given the author of the best unpublished contribution on proctology or allied subjects. Competition is open to all physicians. Information may be secured from the Academy, 43-55 Kissena Boulevard, Flushing, New York.

The annual convention of the National Society for Crippled Children and Adults will be held at the Hotel Statler, Boston, November 3-5.

The 19th annual congress of the United States and Canadian Sections of the International College of Surgeons will be held in Chicago, with headquarters at the Palmer House, September 7-10. Information may be obtained from the College, 1516 Lake Shore Drive, Chicago 10, Illinois.

The Council on Postgraduate Medical Education of the American College of Chest Physicians, in cooperation with the respective state chapters of the college as well as the staffs and faculties of the local hospitals and medical schools, will sponsor the 9th Annual Postgraduate Course in Diseases of the Chest at the Hotel Knickerbocker, Chicago, October 18-22. A similar course at the Hotel New Yorker, New York City, will be November 8-12. Tuition for each course is $75.

Details may be obtained from the College, 112 East Chestnut Street, Chicago 11, Illinois.

Schools of professional nursing in the United States admitted 43,327 new students in 1953, an increase of 1.8 per cent over 1952, it was announced recently by the Committee on Careers, National League for Nursing. Included in the group are students from three-year diploma programs and the four- and five-year collegiate programs.

In Kansas, where there are 26 schools of nursing, 641 students were admitted in 1952 and 604 in 1953.

All products advertised in the JOURNAL have been approved by the various councils of the American Medical Association.

In 1953, 21,400,000 Americans were injured in traffic accidents.

DEATH NOTICES

RICHARD G. MEISBURGER, M.D.

Dr. Richard G. Meisburger, 44, anesthesiologist at Bethany Hospital, Kansas City, died on May 26 after suffering a heart attack. A graduate of the University of Kansas School of Medicine in 1940, Dr. Meisburger interned at St. Luke's Hospital and was resident anesthesiologist at General Hospital, Kansas City, Missouri, before he was called into service in the Navy during World War II. He was an active member of the Wyandotte County Medical Society and of the Kansas City Society of Anesthesiologists. He was also a lecturer at the Kansas City University School of Dentistry.

CLARENCE E. BOUDREAU, M.D.

Dr. C. E. Boudreau, 76, El Dorado, died on June 14 as the result of injuries suffered in an automobile accident the day before. His first medical work was done at the Fort Worth School of Medicine, and he later studied at Columbia University College of Physicians and Surgeons, New York, from which he was graduated in 1913. He received his Kansas license the same year, and during his years of practice he specialized in surgery. He was a member of the Butler County Medical Society and a fellow of the American College of Surgeons.

CLASSIFIED ADVERTISEMENTS

FOR SALE—X-ray equipment; complete Profexray, 100 MA 100 KVP, double tube unit. None better made, used very little, practically new, save $1,000. Write the Journal 11-54.

WANTED: Dermatologist for seven-man group in Midwestern city. Above average facilities with excellent financial opportunity. Write the Journal 13-54.

FOR SALE—Office equipment, Picker Duo-column portable x-ray, diathermy, lamps, instruments, medical books and other equipment. Very reasonable prices. Write the Journal 14-54.

WANTED: Pediatrician for seven-man group in Midwestern city. Above average facilities with excellent financial opportunity. Write the Journal 12-54.

Professional Films

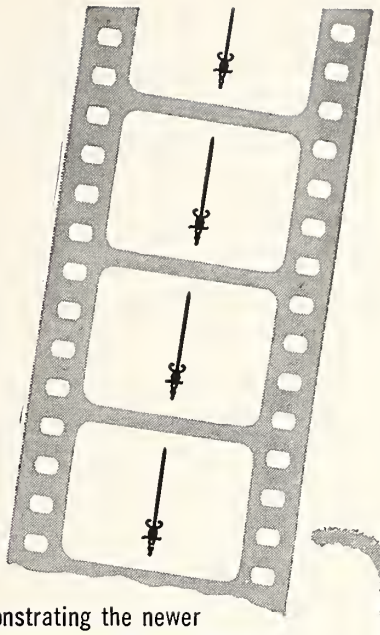
for

Hospital Staff Conferences

Medical Schools

Postgraduate Refresher Courses

State and County
Medical Society Meetings



A distinguished series of color films graphically demonstrating the newer diagnostic techniques in cancer. Sponsored jointly by the American Cancer Society and the National Cancer Institute of the United States Public Health Service.

Cancer — The Problem of Early Diagnosis

Breast Cancer — The Problem of Early Diagnosis

Gastrointestinal Cancer — The Problem of Early Diagnosis

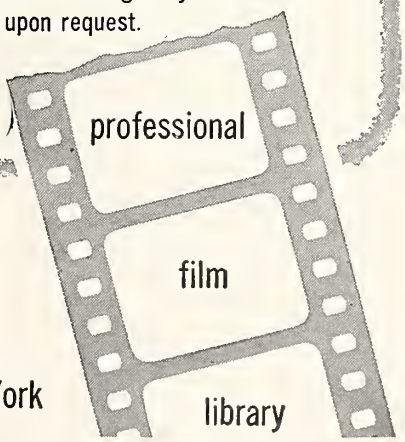
Uterine Cancer — The Problem of Early Diagnosis

Oral Cancer — The Problem of Early Diagnosis

Lung Cancer — The Problem of Early Diagnosis (In production for winter release)

All are 16 mm. sound films in color

As a service to the medical profession, showings of these and other teaching films in our Professional Film Loan Library will be arranged by the Division of the American Cancer Society in your state upon request.



American Cancer Society, inc.

47 Beaver Street, New York 4, New York

Gamma Globulin Provided by Red Cross

Nearly 5,000,000 cubic centimeters of gamma globulin have been provided to the American people by the Red Cross since the Office of Defense Mobilization made an emergency request for the serum a year ago. An additional 5,000,000 cubic centimeters will be ready by April, and a million more by the close of the year.

Of the amount distributed so far, 2,930,818 cubic centimeters went to state health officials for use during the polio season. The balance, added to that being produced now, will provide a substantial supply for use in 1954.

A survey made recently for the *Journal of the American Dietetic Association* reports that Kansas physicians are making good use of material supplied by that group for simplifying and standardizing the diet of diabetic persons. Kansas was among the 10 states ranking highest in the list of those using the material.

The diet information was compiled from studies made jointly by the American Diabetes Association, the American Dietetic Association, and the Diabetes Branch of the U. S. Public Health Service.

In 1953, 38,500 Americans were killed in traffic accidents.

★ PETRO'S SURGICAL APPLIANCES ★

Surgical Support Fittings by Trained Technicians.

Since 1930, Quality Appliances for Men, Women and Children.

- Abdominal Supports
- Back Supports
- Bras—Breast Forms
- Elastic Hosiery
- Knee Braces
- Ankle Braces
- Taylor Braces
- Trusses

"All Fittings Subject to the Doctor's Approval"

618-20 Quincy TOPEKA, KANSAS Phone 40207

It's Convenient to Buy Kanox

THERAPEUTIC OXYGEN AND ANESTHETIC GASES

Oxygen Therapy
Regulators and Equipment
Repair Service for
Regulators, Flowmeters
and Humidifiers



Nitrous Oxide
Ethylene
Carbon Dioxide
Helium
Oxygen
Cyclopropane

KANSAS OXYGEN, Inc.

Phone 5-5551 HUTCHINSON, KANSAS Box 551

NOT ARTHRITIS BUT ARTHRALGIA...

If the patient complaining of aching joints is a woman between 37 and 54 years of age, it is highly possible that she is suffering from arthralgia rather than arthritis.[1] It has been estimated that arthralgia occurs in about 40 per cent of women with estrogen deficiency, and is exceeded in frequency only by symptoms of emotional or vasomotor origin.[2] In fact, arthralgia may be as indicative of declining ovarian function as the classic menopausal hot flushes.

Arthralgia, however, is just one of a vast number of distressing but ill-defined symptoms that may be precipitated by the loss of estrogen as a "metabolic regulator." Other good examples are insomnia, headache, easy fatigability, and tachypnea.

Because these symptoms sometimes occur years before or even long after cessation of menstruation, they are not always readily associated with estrogen deficiency, and the tendency may be to treat them with medications other than estrogen. Obviously, sedatives and other palliatives cannot be expected to produce a satisfactory response if an estrogen deficiency exists. Only estrogen replacement therapy will correct the basic cause of the disorder.

"Premarin" is an excellent preparation for the replacement of body estrogen. In "Premarin" all components of the complete equine estrogen-complex are meticulously preserved in their natural form. "Premarin" produces not only prompt symptomatic relief but a distinctive "sense of well-being" which is most gratifying to the patient.

1. Greenblatt, R. B., and Kupperman, H. S.: M. Clin. North America *30*:576 (May) 1946. 2. McGavack, T. H., in Goldzieher, M. A., and Goldzieher, J. W.: Endocrine Treatment in General Practice, New York, Springer Publishing Company, Inc., 1953, p. 225.

"PREMARIN"®

ACCEPTED COUNCIL ON PHARMACY AND CHEMISTRY AMERICAN MEDICAL ASSOCIATION

Estrogenic substances (water-soluble) also known as conjugated estrogens (equine)
Available in tablet and liquid form
has no odor . . . imparts no odor

NEW YORK, N. Y. MONTREAL, CANADA

5410

It seems apparent that the general practitioner is going to be involved in the actual treatment of the tuberculosis patient to a much greater extent than he has in the recent past and fewer patients will be treated by specialists in chest diseases. Another factor acting in the same direction is the relatively shorter time that the average patient will spend in a hospital and the fact that such hospitalization is less likely to be in a remote institution. The family physician, therefore, will be less likely to lose supervision of his patient. This trend requires a change in the nature and emphasis of postgraduate medical conferences and courses of instruction, which must be directed more toward the general practitioner.—*James E. Perkins, M.D., NTA Bulletin, May, 1954.*

Among patients admitted to general hospitals, the prevalence of definite and suspected tuberculosis and of probably active tuberculosis is twice as high as among individuals x-rayed in mass surveys. Thus 14 (1.4 per cent) of every 1,000 patients show on admission x-ray the presence of definite or suspected tuberculosis as against 7 for every 1,000 in mass surveys. Two of every 1,000 x-rayed on admission to general hospitals show probably active tuberculosis as against one for mass surveys.—*1952 Annual Report, N.Y.S. Department of Health.*

THE LATTIMORE-FINK LABORATORIES

Topeka — El Dorado
Kansas

J. L. Lattimore, A.B., M.D., Pathologist
A. A. Fink, A.B., M.D., Pathologist
H. C. Ebendorf, M.T., Serologist
A. C. Keith, B.S., Chemist
L. W. Hull, A.B., Bacteriologist
Walter Norris, A.B., Chemist

Anatomical and Clinical Pathology

Containers Furnished Upon Request



Woodcroft Hospital--Pueblo, Colorado

A private hospital for the scientific treatment of neuro-psychiatric disorders, including alcoholism and drug addiction. Beautiful landscaping and home-like surroundings afford a restful atmosphere. Accommodations vary from single rooms with or without bath to rooms en suite, allowing for segregation of guests.

Detailed information furnished on request.

KARL J. WAGGENER, M.D.

people need a doctor?"

We're telling the millions of readers of LIFE, TIME,

Saturday Evening POST, NEWSWEEK, and TODAY'S HEALTH

—"All of them!"

The message shown on the opposite page is the latest advertisement in Parke, Davis & Company's "See Your Doctor" campaign which has been continuously published for the past 26 years.

We believe it a part of our responsibility as a maker of medicines to point out to the general public that the doctor is the best "preventive medicine" a family can have.

To be of real service to the cause of Medicine, our messages must not only be given wide circulation but must be the type that people will find interesting and readable. So we try hard to make the general subject of *prompt and proper medical care* "come alive" to the man on the street, the woman in the home.

Seven of these messages are reprinted in the booklet, "Your Doctor and You." If you wish a few copies for your reception room table, please let us know.



PARKE, DAVIS & COMPANY

Research and Manufacturing Laboratories, Detroit, Michigan

TABLE OF CONTENTS

AUGUST, 1954

Original Articles

Treatment of the Acute Alcoholic from a General Practitioner's Viewpoint—Z. Miles Nason, M.D., Kansas City, Kansas . . . 436

Treatment and Management of Alcoholics in a General Hospital—Thomas L. Foster, M.D., Halstead, Kansas 441

Treatment of the Alcoholic Patient in Kansas State Mental Institutions—George W. Jackson, M.D., Topeka, Kansas 446

Alcoholics Anonymous—Mr. X. 451

Sodium Pentothal Solution, an Adjuvant in the Treatment of Acute Alcoholism—DeMerle E. Eckart, M.D., Hutchinson, Kansas . . 453

Editorials

Approach to Problems of Alcoholism . . . 457

Federal Health Programs—Part II . . . 457

Vaccine Evaluation Phase 458

Miscellaneous

The Management of Incurable Cancer—Tumor Conference 460

Blue Shield 472

Physiological Theories of the Etiology of Alcoholism—Senior Thesis 474

Entered as second-class matter at the post office at Fulton, Missouri, June 19, 1953, under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of October 3, 1917, authorized on July 2, 1918. Office of Publication: 1201-05 Bluff Street, Fulton, Missouri.

THE JOURNAL OF THE KANSAS MEDICAL SOCIETY

Editorial Board

Orville R. Clark, M.D., *Editor,* Topeka

John W. Cavanaugh, M.D., Topeka

David E. Gray, M.D., Topeka

Richard Greer, M.D., Topeka

Dwight Lawson, M.D., Topeka

Associate Editors

Donald P. Trees, M.D., Wichita

Glen R. Shepherd, M.D., Kansas City

Information

Ownership: The Journal is a non-profit publication owned and published monthly by the Kansas Medical Society.

Subscription: A year's subscription to the Journal is included in membership in the Kansas Medical Society, with $2.00 of each member's dues apportioned to the Journal. Rates to others, except in foreign countries, $4.00 per year or 60c per copy.

Material: Scientific articles, editorials, and data of general interest are invited from all members. Articles are to be submitted on condition that tbey are contributed solely to this publication. A rigbt is reserved to reject any material deemed unsatisfactory.

Manuscripts: Only manuscripts that are typewritten on one side, double spaced, and original copies can be accepted. Manuscripts will be returned upon request.

Advertising: All advertising contracts, and all copy from advertisers under contract are subject to approval of the editorial board. Copy should be received by the 20th of the month immediately preceding the month of publication.

Non-Responsibility: Although an effort is made to publish only accurate articles and legitimate advertisements. the Journal denies legal responsibility for any statements, opinions, or advertisements appearing under the names of contributors or concerns.

Pauline Farrell, Managing Editor
and Advertising Manager

Oliver E. Ebel, Business Manager

THE KANSAS MEDICAL SOCIETY

President . . . . . Murray C. Eddy, M.D., Hays

Past President . . . . . Lucien R. Pyle, M.D., Topeka

President-Elect . . . . . John M. Porter, M.D., Concordia

First Vice-President . . . Clyde W. Miller, M.D., Wichita

Second Vice-President, Conrad M. Barnes, M.D., Seneca

Constitutional Secretary, James A. Butin, M.D., Chanute

Treasurer . . . . . John L. Lattimore, M.D., Topeka

A.M.A. Delegate, 1954-1955, George F. Gsell, M.D., Wichita

A.M.A. Delegate, 1955-1956, Laurence S. Nelson, M.D., Salina

Councilors and Councilor Districts

First District, Frederick E. Wrightman, M.D., Sabetha

Second District . . . Glenn R. Peters, M.D., Kansas City

Third District . . . . . H. Penfield Jones, M.D., Lawrence

Fourth District . . . . Charles E. Vestle, M.D., Humboldt

Fifth District, Severt A. Anderson, M.D., Clay Center

Sixth District . . . . . Floyd C. Taggart, M.D., Topeka

Seventh District . . . . . To be announced

Eighth District . . . . . James E. Hill, M.D., Arkansas City

Ninth District . . . . Laurence S. Nelson, Jr., M.D., Salina

Tenth District . . . . . Harold M. Glover, M.D., Newton

Eleventh District . . . . Norton L. Francis, M.D., Wichita

Twelfth District . . . . . Cyril V. Black, M.D., Pratt

Thirteenth District . . Lloyd W. Reynolds, M.D., Hays

Fourteenth District . . . . Justin A. Blount, M.D., Larned

Fifteenth District . . . . Robert G. Klein, M.D., Dodge City

Sixteenth District . . . . . James L. Jenson, M.D., Colby

Seventeenth District . . . . . H. Preston Palmer, M.D., Scott City

Oliver E. Ebel, Executive Secretary
Rueben M. Dalbec, Executive Assistant

Executive Office: 315 West Fourth Street, Topeka

THE JOURNAL
of the
KANSAS MEDICAL SOCIETY

Copyright, 1954, by The Kansas Medical Society

Volume LV AUGUST, 1954 No. 8

Helpful Facts for the Physician

It should be every physician's responsibility to accept a major role in the improvement of health throughout his own community. This is true regarding individual specific diseases, and it is equally important when the state attempts some type of progressive coordinated public health movement.

Alcoholism must be considered a disease and, as such, it presents itself for treatment in the office of every doctor in Kansas. With the advent of a State Commission on Alcoholism, The Kansas Medical Society will be asked to assist in setting up a state-wide program of education and better understanding, such measures eventually making their appearance in each individual doctor's office and each community hospital where the main responsibilities for treatment rightfully exist.

A physician naturally has his likes and dislikes, and he may turn away from any alcohol problem just as some shy away from other specific medical or surgical requests for therapy. The family physician should take the same attitude toward handling cases of alcoholism that he takes toward management of any other disease. First of all, he must recognize the problem, initiate proper therapy himself, or see that the patient is referred through adequate channels.

It is to be expected, and rightfully so, that the citizens of Kansas look to their local physicians and hospitals for treatment of the great majority of their illnesses. Individual communities are constantly expanding and augmenting their facilities. In these local projects the family physician must be an important part of the organization.

Since alcoholism is essentially the symptom of a rather complicated illness, the correction must involve the efforts of a team. The members of this team probably vary from case to case. However, the family physician, as well as the medical consultant, will be in most cases a deciding influence.

The physician who directs therapy for patients suffering from alcoholism must intelligently prescribe vitamins and drugs, and, above all, he must provide a sympathetic, understanding, reassuring atmosphere for recovery and rehabilitation.

KARL E. VOLDENG, M.D., *Chairman*
Kansas State Commission on Alcoholism

Treatment of the Acute Alcoholic from a General Practitioner's Viewpoint

Z. Miles Nason, M.D.
Kansas City, Kansas

Alcoholism has been presented by many present day writers as the fourth major health problem in the United States. Though there is no concrete evidence the disease is increasing or decreasing, it has been a serious one throughout recorded history of our civilization. In the United States there are an estimated 65,000,000 people who partake of alcoholic beverages; there are about 4,000,000 problem drinkers and about 75,000 confirmed alcoholics.[1] These figures are of even greater significance when compared with the 500,000 cases of known tuberculosis.[2]

Alcoholism is a disease in which an uncontrolled excessive amount of alcoholic beverages is ingested, either regularly or periodically. Tiebout states that alcoholism is a symptom which has taken on disease significance. He further states that, starting as a symptom of underlying factors, it gains momentum until it gets out of hand and becomes a disease in itself.[2]

Tiebout reasons that if the treatment of addictive drinking depended solely on discovering underlying factors and eliminating them, it should be possible for the alcoholic to drink normally after the excision of these factors. All evidence points to the fact that the alcoholic cannot do this. He further emphasizes the existing confusion in attitudes toward alcoholism. Is it in fact a symptom or a disease? If one recognizes the existence of underlying factors in alcoholism, then addictive drinking itself is symptomatic of the disease, which has given rise to abnormal behavior.

We should expect that once a predisposing factor, for instance metabolic disturbance, and alcohol were brought together, an addiction soon would be formed. That this is not true is evidenced by the fact that vast majorities of alcoholics show a development of abnormal drinking patterns taking place over a period of years, which would indicate that the relationship between drinking and the hypothesized predisposing characteristics is of central importance; thus, personality must play a very important factor. It may be that the terrain on which alcoholic addiction may or may not grow is not sufficient cause of addiction alone; certain forces must act on the terrain to bring about addiction or abnormal drinking.

Ullman[3] considers addictive drinking as resembling a symptom but not necessarily a symptom of personality or other disorder. He describes it as a tension reducing activity, with the source of tension lying in the ordinary problems of human beings. These kinds of tension are of the same character that all human beings face while living in a complex system of relationships with others of their kind. Therefore, addictive drinking is symptomatic of the existence of tension, but the pathology lies in the fact that the particular response exhibited in alcoholism is practiced in excess, and it obviates the evocation of other more appropriate responses which might alter the realities of the problem situation.

Alcoholism is a progressive ailment which is not diagnosed until the ingestion of alcohol becomes so great that there is a definite interference with work efficiency, disturbances in home and in social life, and a complete change in the personality of the individual. The fundamental point of distinction, which all alcoholics have in common, is that they cannot drink in a normal manner. This is functional evidence that there is something amiss in either the mental or physiological condition in the alcoholic.[4] Stated in another way, alcoholism is a symptom syndrome of a disease process.

Tiebout[2] points out that although drinking to excess may have started out as a symptom of some underlying factor, it gains momentum until it gets out of hand and becomes a disease in itself. In support of this view he states that no amount of probing or unraveling of basic personality conflicts will ever allow the alcoholic to return to normal drinking. Once alcoholism is definitely established, it seems that even with removal of all known conflicts, it is not possible for the alcoholic ever to indulge in controlled drinking. Thus, alcoholic excess cannot be interpreted as a means of escaping reality, but rather is characterized by a compulsive type of drinking. Compulsion is psychiatric terminology, referring to a form of mental pathology in which the individual is impelled to do things against his will and judgment.[5]

This compulsive quality of drinking found in alcoholism has been a source of great discussion and the cause of a great deal of confusion. Recent investigation has helped to explain why various attempts to treat alcoholism by psychoanalysis have not been successful. Psychotherapy, which does not in-

Member, Kansas Commission on Alcoholism; Consulting Physician, New Mexico Alcoholic Commission.

corporate some manipulation of the daily activities of the alcoholic, has apparently yielded poor rates of success.

In the case of Alcoholics Anonymous, perhaps the most effective form of psychotherapy known, there exists a combination of treatment factors which the experience of A.A. indicates must be combined for the maintenance of sobriety in its members. A member of A.A. is involved in religious or spiritual activities in group psychotherapy, in the activities of helping the newer alcoholic, and also in a number of recreational activities with other members. Most significant, however, is that the various factors are arranged in a 24-hour program of activity. The alcoholic in A.A. is doing things, some of which may take on a ritualistic significance, thus the emphasis is placed on tension-reducing activity rather than drinking. The addictive drinker is introduced, or rather re-introduced, to socially accepted reactions to tension. Possibly the reason for such poor results in young alcoholics is that the response pattern to which he might regress is often not appropriate to adult life and, therefore, his retraining and conditioning is more difficult.

Ullman[3] states that successful therapy of alcoholics demands a development of substitute tension-reducing responses. The manner in which they are produced is by making such changes in the way of life of an alcoholic that he has an opportunity to form habit patterns which will be satisfactory in terms of coping with psychic tensions, but which will enable the individual to deal with the realities of situations rather than anesthetize himself against them.

Lowell and Tintera[6] state that there is a functional metabolic defect which can give rise to alcoholism, which may develop as a result of excessive drinking. This defect is hypoadrenalcorticism. They believe this may be compared with subclinical Addisonism. Stress, worry, grief, anxiety, or fear may act as alarming stimuli, creating a functional defect of the adrenal cortex, producing hypoadrenalcorticism. This condition, whether constitutional or acquired, may contribute substantially to an increase in nervous symptoms, ranging from irritability to depression, negativism, and other personality changes associated with alcoholism. They postulate a vicious cycle of glandular deficiency. Worry or any of the strong emotions will further the deficiency. Imposing the action of alcohol on such a condition, depressing the adrenal ketosteroids further, causes an initially bad situation to become worse.

This concept of Lowell and Tintera is based on their observation of 1000 cases of alcoholism, in which they found a relatively low blood sugar level (60 mg.%). A glucose tolerance curve which usually started at this low level rose more rapidly than normal to a moderate hyperglycemic level, remaining there in the form of a plateau, and then precipitiously dropping to the starting level. They also report a relative lymphocytosis and eosinophilia associated with more than half the cases studied. These findings are indicative of a deficiency in the adrenal cortex.

The reason that this glandular dyscrasia can contribute to alcoholism is that with the drop in blood sugar, there is an onset of extreme nervousness and weakness. The alcoholic quickly learns to combat this weakness and nervousness with alcohol, which gives a transient relief but quickly raises blood glucose level. This transient relief is followed by exacerbation of symptoms, and the alcoholic again responds by taking more whiskey to guard against this let-down. This is repeated over and over again, and the alcoholic becomes inebriated. They point out further that these hypoglycemic levels can also occur spontaneously or following a meal rich in carbohydrates but low in protein and low in other lasting foods. This, perhaps, might be the explanation for some of the relapses which occur in alcoholics after they have maintained long periods of sobriety.

J. J. Smith[7] believes that a person is alcoholic because of a biogenic defect in the sense of a hereditary pattern of metabolic disturbances. He believes that alcoholic behavior is a manifestation of personality maladjustment to both external and internal stresses and that the metabolic defect is primarily a pituitary insufficiency, to which adrenal cortex inactivity is secondary. He has reached this conclusion after intensive biochemical investigation of this disease. In many of his studies, deficiency of both adrenal cortex and hypophysial functions was found. As evidence of this, he cites a fasting hypoglycemia which is often found in alcoholics, hyper-insulin glucose tolerance curves, and a large number of alcoholics who have shown abnormal water tolerance tests and many who show low serum sodium and chloride levels.

He found, however, although the 17-ketosteroid levels were quite low following delirium tremens and in patients suffering from severe peripheral neuropathy, that in 100 other alcoholic patients the 17-ketosteroids were on the average only slightly below the normal level. Another interesting finding by him was that ascorbic acid, which is necessary for adequate adrenal function, is usually at a low plasma level in the alcoholic. In experiments with normal rats, he found that alcohol in sufficient quantities to produce mild intoxication depleted the adrenal cortex of cholesterol and ascorbic acid. This was not seen in hypophysectomized rats. It was also found that quantities of alcohol which were tranquilized in normal rats killed the hypophysectomized animals. It is to be concluded, therefore, that alcohol stimulated the adrenal cortex by way of the pituitary.

He further showed that in alcoholics the eosin-

ophil fall following the administration of ACTH was normal, but that this response was poor following stimulation of the adrenal by way of the pituitary with adrenalin and alcohol. Thus he concluded that the alcoholic has an adequate adrenal cortex reserve, but the pituitary stimulation is insufficient for normal glandular functioning. This group of investigators believes that alcoholism is therefore anteceded by and induced by endocrine dyscrasias.

Roger J. Williams[8] believes that alcoholism is a genetotrophic disease, stemming from both an inborn trait and a nutritional deficiency. Based on his experimental work with rats, he believes that a craving for alcohol can be caused by mild nutritional deficiencies in any of the several essential accessory food substances. He believes that elimination of these deficiencies will cause elimination of the craving for alcohol by the alcoholic and permit his return to normal drinking. This has not been proved by subsequent investigations. Relatively high incidence of vitamin deficiency is unquestionably present in this disease, in that the alcoholic substitutes alcohol for food, and alcohol certainly makes a diet that is poorly balanced and deficient in vitamins. Multiple peripheral neuropathy, alcoholic beriberi, and alcoholic pellagra are three well defined deficiency diseases seen in alcoholism. Among the mental disorders in which vitamin deficiencies play an important role are Wernike's syndrome, nicotinic acid deficiency encephalopathy, delirium tremens, and Korsakoff's psychosis.[9]

In view of the above facts, it is apparent that the alcoholic is an acutely ill individual, suffering from multiple vitamin deficiencies and a disturbed cellular environment due to endocrine imbalances, with a difficult mental and physiological handicap to overcome before he can hope to recover. He definitely must have assistance if he hopes to recover, and since the general practitioner is the medical man to whom he first appeals, it is important that the practitioner give sympathetic understanding and intelligent direction and treatment.

The sobering up of the acutely intoxicated alcoholic is usually erroneously regarded as a minor problem. It is not generally realized that patients in this condition, if not treated effectively with measures both adequate and humane, may develop severe physical and mental complications. The number of alcoholics found dead, often of unknown or ill defined causes, in jails, charity hospitals, hotel rooms, and alleys, will bear witness to the seriousness of this problem.[10]

Examination of textbooks and periodicals reveals a wide variety of methods of treating acute alcoholism. It is indicative of the importance of the problem that none of the procedures suggested have been too successful. Either they are not acceptable to the alcoholic, do not give rapid relief of painful symptoms, or do not establish a good rapport between the patient and the physician, which is essential if permanent sobriety may be achieved.

In the treatment of alcoholism, one must remember that it is an addiction disease. Therefore, drugs which produce or tend to produce addiction should be avoided. Opiates and barbiturates are addiction drugs, and the indiscriminate use of such drugs has produced morphinism and barbituratism in the alcoholic.[11] The narcotizing effect of opiates and barbiturates is much greater in the alcoholic than in the so-called normal individual and much more lasting. The only safe narcotizing drug used in treatment of this condition is paraldehyde, and it is used only in a case of severe delirium tremens when all other measures fail. Recent research into the role of hormone dyscrasias and the developing of the concept of replacement therapy to restore normal homeostasis in diseased patients has done much to simplify and greatly enhance recovery of patients suffering from alcoholism.

Before beginning to treat the acutely sick and drunk alcoholic, the physician should call to mind a few primary psychological concepts. Alcoholics are filled with self-condemnation and therefore resent any form of lecture as to their wrong doings. They are suspicious and afraid of chemical cures, and no treatment should be initiated without assurance that they will not be made to suffer more, that the treatment will not harm them, and that the object of the treatment is to help them get well. Such assurance must impress them that the treatment is not only to help them obtain sobriety, but also to aid them in changing their mental attitudes so that they may maintain continuous sobriety. In talking to them it is necessary to remember that their mental state is greatly fogged, and therefore much repetition is required for them to grasp an idea; that their thought and reflex processes are greatly retarded; and that no attempt should be made to hurry them as this irritates them and generates rebellion on their part toward any person forcing them.

Alcoholics are not drunks by choice; their chief objective has been the exact opposite. Their greatest ambition has been to learn how to handle their liquor, in other words, to become a controlled drinker. Throughout the treatment, they should be impressed with the fact that their nervous systems have become allergic to alcohol and that they will never again be able to drink in a controlled manner. Any further attempt to drink will rapidly place them in the same deplorable condition that they were in when they appealed for help and treatment. The following is a method of treatment which has been used in 1500 cases of alcoholism during the past four years with surprising rapidity of recovery.

BASIC TREATMENT FOR THE ACUTE ALCOHOLIC

If the patient, when first seen, is obviously quite drunk, no treatment should be started until he begins to clear mentally, develops a coarse tremor, and clearly begins to show the need for sedation. At that time 3 cc. of Lipo-Adrenal Cortex* intramuscularly and 1 cc. of B-1 (100 mg.) and 1 cc. of B-Complex with C should be given either intravenously or intramuscularly. Six hours after the first injection, 1 cc. of Lipo-Adrenal and 1 cc. of B-1 and 1 cc. of B-Complex with C should be given intramuscularly. Twelve hours after the second injections, the dosage of 1 cc. of Lipo-Adrenal and 1 cc. of B-1 and 1 cc. of B-Complex with C is repeated. Concurrently, throughout the treatment, give 1 teaspoon of table salt in six ounces of water with first injections and then repeat every 3 hours for a total of five doses.[13] If there is a history of partial barbiturate addiction, one may double the salt dosage to a total of 10 doses administered at intervals of 3 hours. If the patient is obviously deeply sedated and respiration is 8 to 12 per minute, or if he appears in rather a deep coma, Metrazol, 2 to 4 cc. given intravenously, often improves the general condition and may be given before initiation of Lipo-Adrenal treatment as outlined above.

If history and observation suggest barbiturate addiction overshadowing alcoholic addiction, then the salt water alone often produces excellent results. However, in such a case, if additional medication appears to be needed, as evidenced by extreme nervousness, 2 or 3 cc. of Lipo-Adrenal given intramuscularly during the course of recovery may produce sufficient sedation to keep the patient from developing tremor. Thus, Lipo-Adrenal, even in the barbiturate addict, often effects relief with a single 2 or 3 cc. dose. It has been found that clinical improvement in all patients is better and more rapid if no food is given for the first 24 hours. After that period of time, a high protein, high salt, low carbohydrate diet appears to aid the patient's recovery, with a subsidence of nervous and mental agitation much more quickly than the high carbohydrate diet formerly so widely used. Tycopan capsules** instigated after the first 24 hours, when food is started, one before each meal, aid substantially in correcting the vitamin deficiency. The addition of black coffee, given ad lib, often alleviates, to a considerable extent, any mental depression present.

If the acute alcoholic patient is suffering from delirium tremens or having convulsions, then the above treatment should be augmented by the administration of adrenalcorticotrophic hormone (ACTH***), in gel form. One cc. (40 U) should be given with each injection of Lipo-Adrenal as outlined in treatment above. If delirium tremens continue without any interval of remission, for the first 12 hours, the dosage of ACTH may be increased to 2 cc. (80 U) doses at an interval of six hours.[13] Then, if the patient is still delirious, no further treatment should be given as he will usually clear after such treatment within 36 to 48 hours.

Three contraindications exist against the administration of ACTH. They are: (1) history of gastric or duodenal ulcer, within the last five years; (2) history of familial insanity, and (3) tuberculosis. The reason for the first is that administration of ACTH has been known to produce a blow-out or perforation of the ulcer. This occurs because ACTH causes an increase in peristaltic action of the muscles of the stomach; it also increases the amount of acidity in the gastric juice. These two factors sometimes produce perforation. The reason for the second contraindication is that ACTH can produce a deep psychosis which may last for several months in a patient whose family history shows insanity. A history of tuberculosis during the past five years is a contraindication because this drug has been known to reactivate this disease.

A locked room with no movable objects is much better than straps or restraints for confining patients in delirium.

In cases of extreme agitation persisting 36 hours or more, where one feels that sedation is imperative because the patient is obviously worn out, 4 drams of paraldehyde may be given in two ounces of whiskey as a single dose. This has been known to produce adequate sedation after ACTH has failed.

In treating women alcoholics, regardless of age, the administration of 5 mg. of aqueous estrone intramuscularly materially assists in producing a smoother course of recovery, with much more rapid subsidence of nervous symptoms. This should be administered at the termination of the adrenal cortex injections. If the patient has a history of menopausal symptoms before her drinking and is in the late 30's or 40's, she should be advised to see her family physician, after discharge, for estrone injections weekly or semi-monthly for the ensuing three months or longer, depending upon what her physician advises. Premenstrual tension which these women often have may produce tensions which can, and sometimes do, precipitate a relapse and the beginning of another alcoholic spree.

For men alcoholics in the late 40's or early 50's, with a history of insomnia, diminished libido, or sexual incompetence, 50 mg. of testosterone propionate will often improve nervous conditions, promote a feeling of well being, and hasten recovery. This also is given at termination of adrenal cortex injections.

* Upjohn & Company.
** Eli Lilly and Company.
*** Armour and Company.

The alcoholics' only hope for permanent sobriety exists in a change of thinking, which is probably most easily obtained through group psychotherapy as practiced by Alcoholics Anonymous. This organization could well be their short cut to peace, contentment, and a happy life. With honest effort on their part in this organization, it is possible for them to achieve a change in their mode of living and ways of thinking which will give them sufficient support to maintain permanent sobriety.

SUMMARY

Alcoholism is a disease characterized by marked physical and mental changes in an individual produced by compulsive drinking of abnormally large amounts of alcoholic beverages, either constantly or periodically. The distinguishing characteristic which sets the alcoholic apart from the heavy drinker is his inability to stop drinking once he begins. This lack of control arises from important physiological changes present in the individual prior to the development of alcoholism, also changes resulting from the prolonged use of alcoholic beverages. These changes are deficiencies in the pituitary-adrenal cortical relationship, multiple vitamin deficiencies resulting from a lack of intake, and increased utilization of both vitamins and hormones during drinking periods.

The disturbed organic and functional changes resulting from prolonged drinking by the alcoholic cause him to be a very ill person, mentally and physically, and he should be treated as such. A method of therapy for the treatment of the acute alcoholic by means of adrenalcortical hormones, sodium chloride, and multiple vitamins is presented for consideration.

The use of ACTH in treatment of alcoholic delirium is outlined.

Probably the best form of psychotherapy available to assist in obtaining permanent abstinence for the alcoholic is that offered by the Alcoholics Anonymous organization.

BIBLIOGRAPHY

1. Block, Marvin A.: Alcoholism—the general practitioner's viewpoint, New York State Journal of Medicine 50:14, 1950.
2. Tiebout, Harry M.: The role of psychiatry in the field of alcoholism, Quarterly Journal of Studies on Alcohol 12:1, 1951.
3. Ulman, Albert D.: Psychological mechanism of alcohol addiction, Quarterly Journal of Studies on Alcohol 13:4, 1952.
4. Osborn, Leslie A.: The clinical approach to alcoholism, Journal of the American Medical Association 143:2, 1950.
5. Tiebout, Harry M.: Some aspects of the problem of alcoholism, New York State Journal of Medicine 50:14, 1950.
6. Lowell, H. W., and Tintera, J. W.: Hypoadrenalcorticism in alcoholism and drug addiction, Geriatrics 6:1-11, 1951.
7. Smith, James J.: The endocrine basis and hormonal therapy of alcoholism, New York State Journal of Medicine 50:14, 1950.
8. Williams, Roger J.: Nutrition and alcoholism, University of Oklahoma Press, Norman, 1951.
9. Lolli, Giorgio: Alcohol Addiction, The American Journal of Nursing 48:8, 1950.
10. Nason, Z. M.: A safe method of detoxicating the acutely ill alcoholic, Quarterly Journal of Studies on Alcohol 8:1 (June) 1947.
11. Sedatives and the alcoholic: Works Publishing, Inc. New York, 1952.
12. Smith, James J.: The treatment of acute alcoholic states with ACTH and adrenocortical hormones, Quarterly Journal of Studies on Alcohol 11:2 (June) 1950.
13. Silkworth, W. D.: Chloride levels in the blood of alcoholic patients in relation to phenomena of craving, Quarterly Journal of Studies on Alcohol 11:3 (Sept.) 1950.
14. Alcoholics Anonymous: Works Publishing Company, New York, 1943.
15. Jellinek, E. M.: Alcohol addiction and chronic alcoholism, New Haven Yale University Press: London Humphrey Milford —Oxford Press 1942.

Since alcoholism is an illness, physicians must approach it as such—not as a crime against society. In differentiating between the heavy drinker and the alcoholic, the most important distinguishing feature is compulsion. Overcoming this compulsive tendency depends upon careful study and treatment—medical, psychiatric, and socio-economic. This last element embraces education, social study, and close collaboration with Alcoholics Anonymous.

Marvin A. Block, M.D.
G. P., September, 1952

Treatment and Management of Alcoholics in a General Hospital

Thomas L. Foster, M.D.

Halstead, Kansas

In order to formulate from first hand information a general idea of the extent of the alcoholic problem in at least one area of Kansas, I consulted the chief of police of one of our Kansas cities that I believe to be representative. The number of "drunks arrested" for 1952 and 1953, when compared to the total population of the city, approximated 25 "drunks arrested" per 1000 population for each year. He also informed me that 44 per cent of these individuals were repeaters.

This is only one segment of that particular city's alcoholic problem. I have no information as to how many individuals were cared for by the local hospitals, Alcoholics Anonymous, Salvation Army, physicians, other agencies, friends or relatives, or assisted by the police force without arrest. The latter group must have been quite large, as the members of this particular police force have the feeling that incarceration of an alcoholic serves no therapeutic purpose, but merely tends to protect society, and the individual, temporarily. Incidentally, for many years there have been no deaths in which alcohol played a major role in this particular city jail.

Projecting such figures onto the entire state of Kansas would certainly be more speculative than statistically accurate, but when one does attempt to do so, the result is an alarming figure of 50,000 cases a year in which, at least under certain circumstances, incarceration might be justifiable as an immediate expediency. A figure such as this explains how 53, or 11 per cent, of the fatal highway accidents in Kansas last year involved drivers under the influence of alcohol.[1]

The fact that during the past 9 years 26 states have created commissions on alcoholism clearly indicates growing public determination to do something about the problem. Even if the above figures should prove to be grossly in error in over-estimating the extent of the problem, it is still obvious that our limited psychiatric hospital facilities are unable to meet the need, and our limited number of psychiatrists and clinical psychologists could not cope with the problem even though they had nothing else to do. Just as in any other matter of health, our first line of defense lies with the general hospital, the general practitioner and other local facilities, and the specialists or special facilities need be reserved for those cases in which there is a definite indication for such services.

Although alcoholism is probably more properly defined as a symptom overlying some basic psychopathology, our justification for using it as a diagnostic term lies in the fact that this symptom itself, directly or indirectly, produces a series of secondary effects of a medical, social, economic, and moral nature. These are frequently more of an apparent problem to society and a more obvious threat to the individual than the primary psychopathology, and alcoholics, in addition to their primary psychopathology, do have certain personality characteristics in common that tend to set them in a class by themselves. Furthermore, the treatment of the primary disorder is usually impossible as long as the use of alcohol persists.

Psychotherapy in some form or other, whether it be "just talking things over" with the family physician or spiritual advisor, group therapy in A.A., individual sessions with a psychiatrist, or some other method or a combination, is a basic essential in the successful treatment of any alcoholic. In some cases, the only practical or even possible way to handle the patient is to attempt to restore the defenses against anxiety which often served the patient well before alcoholism developed. It is better to reinforce these defenses than to attempt insight therapy with the hope of bringing about any great personality structural change. Others can benefit materially from individual psychotherapy, and occasionally a complete analysis is indicated. The latter are time consuming and expensive procedures that could not possibly be applied to every case, even if all alcoholics were suitable subjects.

In dealing with the alcoholic, even the experienced psychotherapist is ever aware of the danger of too much probing into the patient's life and emotional problems, stirring up more than he can handle, and he is even more cautious of offering too many interpretations that the patient may misunderstand or misuse.

Our social thinking is gradually getting away from the old concepts that alcohol per se is the basis of the alcoholic's problem, and that it is all a matter of

Halstead Hospital, Hertzler Clinic and Research Foundation, Halstead, Kansas.

"will power" that has caused the alcoholic to be treated with so much contempt, neglect, derision, and ineffective forms of punishment for so long. The amount of research that has been done in the field of alcoholism is woefully inadequate, compared to that which has been done in other medical areas, and this situation is undoubtedly, to a great extent, due to society's moralistic attitude toward the problem.

In order for a general hospital to successfully treat alcoholics, the patient must be viewed in an entirely objective and realistic manner. Most alcoholics, even though quite intoxicated, are quick to recognize anything the least bit contemptuous or punitive on the part of the staff, and such attitudes usually lead to failure as far as therapy is concerned. The attitude of self assurance that the alcoholic so frequently displays must be recognized as merely a shell or a cover-up for a deeply anxietous individual who has learned to use alcohol, at least in part, to allay his anxieties and tensions. It must also be accepted that any direct attack upon this self assurance which would be a direct "loss of face" is absolutely intolerable and frequently leads to immediate return to drinking. The fear of death or disease is certainly not much of a deterrent to the use of alcohol. One group studied[2] revealed 11 per cent as actual suicides, and 28 per cent died directly or indirectly as a result of alcohol.

Alcoholics in general handle their resentments and hostile or aggressive feelings poorly with inadequate normal outlets and, for that reason, inappropriate displays are certainly easily precipitated.

No general hospital wishes to become a place for alcoholics to sober up and get into condition to resume their drinking, thus aiding and abetting the process; neither do they want to accept alcoholic patients whom they are incapable of handling. The selection of cases, along with the attitude of the hospital, may determine the success or failure of any treatment program.

The evaluation of treatability in the case of an alcoholic is similar to that of any other disorder. The more adequate the individual's adjustment to society, prior to the onset of the drinking, the more likelihood there is to restore the individual to his previous adjustment or, in some cases, improve on the original. The basic underlying personality structure has a great deal to do with the prognosis.

The very immature, unstable person whose behavior disturbances date back to early life, including that group often referred to as "psychopaths," who have started excessive drinking early in life, generally offer a poor prognosis. We do quite frequently see schizoid individuals (even borderline schizophrenic persons) who have shown a poor adjustment in certain areas of the total situation but have been fairly successful economically, actually show great improvement in the total personality picture after a short hospitalization and identification with Alcoholics Anonymous. The drinking in such a person usually starts at a later period in life, and we frequently find long periods of abstinence interspersed with periods of excessive alcoholism. Although one must consider the diagnostic pattern of the basic personality, one is really more interested in the individual's adjustment pattern than he is in any one categorical diagnostic entity.

The classification of alcoholics offered by Bowman and Jellinek[3] is probably the most practical one we have. A large percentage of alcoholics fall into the category of secondary addiction. This individual usually gives a history of a fairly adequate adjustment in early life. He usually goes through a period of social drinking which gradually becomes heavier, he depends more and more upon alcohol through emotionally tense periods or situational crises, and finally finds alcohol a necessary part of his environment. He can no longer control his drinking and frequently gets to the place where he has the tell-tale "morning shot." This individual has many personality assets which can be relied upon, and, aside from his alcoholic life, has been a pretty capable person. The general hospital should have a high percentage of success with this type of individual. When these individuals have difficulty maintaining sobriety or experience nervous symptoms while maintaining it, they usually accept psychiatric referral quite well and make excellent patients with a minimum of help.

There are two other groups of alcoholics that have been described in which the prognosis is not so good. One is the symptomatic drinker who has a severe underlying neurosis or even a psychosis, and drinking is only one of many symptoms. This type of individual may need treatment in a psychiatric hospital.

Another type of alcoholic about which we know very little is usually referred to as the "essential alcoholic"[4] or the "addictive alcoholic."[3] This individual launches on a career of severe alcoholism early in life. As a rule his adjustment even without alcohol has never been really satisfactory. Attempts to interrupt the drinking are usually quite turbulent and purely temporary. These are the individuals who frequently go on to the development of Korsakoff's psychosis or some of the other chronic organic deteriorated conditions frequently associated with alcohol.

The diagnostic differences between these types of alcoholics are not always clear-cut. Consequently, in any case we must depend not only on the patient's past history but also to a great extent on the individual's motivation and a sincere desire for help. This can only be determined by evaluation of the total situation. Few alcoholics present themselves for treatment without a certain amount of coercion or

pressure of some type. This pressure may come from many different sources, and we must carefully evaluate how much of a genuine desire it creates in the individual.

There is some truth in the old saying, "The alcoholic must hit what is bottom for him before he can come up." It is only reasonable that no individual would give up his drinking as long as it is serving a purpose, or at least as long as he thinks it is serving a purpose and he sees no great need to discontinue. Undoubtedly many alcoholics have entered a hospital or taken some "cure"—not because they saw any need of it at all but because pressure at home was just too great and compliance was the easiest way to "get them off my neck." Of course, the individual would return to his drinking as soon as he was dismissed, and then he had a counter argument in the form of "Well, I took your old cure and it did not do any good."

Many social drinkers have discontinued the practice because those about them objected, but a more intense motivation than this is required by the genuine alcoholic. He must completely accept the idea that alcohol has become his master, and it is up to him to find a way out. Most alcoholics need outside help. Occasionally we will find a fairly heavy drinker even in the group of secondary addicts who has sufficient insight and adequate personality resources to discontinue the use of alcohol by himself. This, of course, lends credence to the "will power" idea. The individuals who have undergone the longest periods of sobriety have, for the most part, been those who sought help from the hospital or other agencies at their own request, often looking very much the worse for wear and tear at the time and frequently quite intoxicated or in a state of severe hangover. In some way or other they manage to get there under their own power and blurt out what could be paraphrased, "I've got to have help—I've just got to get away from this thing that is ruining me."

Once the alcoholic is admitted to the general hospital, the first problem in many cases is that of acute intoxication which is a self limited condition, but the oxidation of alcohol can probably be hastened by the administration of oxygen. The method of Davis and Robertson[5] consists of the administration of pure oxygen by mask for 20 minutes out of every hour for the first 6 hours or, in case of a psychotic episode or delirium tremens, it may be carried on for 24 to 48 hours.

A complete medical examination and indicated laboratory procedures are, of course, necessary in all cases. In practically every case of severe alcoholism there are some nutritional and metabolic factors that require attention. The alcoholic is almost invariably dehydrated and deficient in salt and in vitamins of the B complex group; usually his protein intake has been inadequate. If he has been drinking heavily, he has had a fairly high caloric intake from his alcohol which, of course, is metabolized as a carbohydrate at the expense of other foods.

One fairly satisfactory plan is to administer intravenously, and as early as possible, 1000 cc. of 10 per cent glucose in saline to which an ampule of one of the parenteral vitamin B complex preparations is added and reinforced with 100 mg. of vitamin B_1, 1000 mg. of nicotinamide and from 50 to 500 mg. of ascorbic acid. Another 1000 cc. of 10 per cent glucose in water may be given 12 hours later without the vitamins. This procedure is usually carried out daily for several days. In individuals with no evidence of renal disorder, one may give 15 gr. of sodium chloride in enteric coated tablets four times a day. Caution is used in elderly individuals who may have circulatory or renal impairment.

The patient is frequently nauseated, certainly has no appetite, and may be unable to handle food. Fresh orange juice is much appreciated by most patients and apparently does a great deal to stimulate an appetite for other foods. Frequently it is the only thing that is really relished by mouth, and it has been my experience that the artificially flavored orange drinks or the dried preparations are not a substitute for either the fresh juice or the concentrated frozen product.

When a sedative is necessary, paraldehyde is probably the sedative of choice. It may be given by mouth or by rectum in two- or three-dram doses. Frequently patients managed as outlined above may get along fairly well without sedation or with just an occasional dose—a disturbed patient may need to have it administered fairly frequently within the first 24 to 48 hours.

The promiscuous use of barbiturates in alcoholics usually leads to disaster by increasing restlessness and confusion and even precipitating a psychosis. Morphine in the alcoholic is a dangerous respiratory depressant.[6] Chloral hydrate has two disadvantages. One, of course, is the fact that chloral hydrate combines with alcohol to produce a toxic product, chloral alcoholate or the well known knock out drops, and of course this chemical reaction could take place in the patient's stomach as well as in the laboratory beaker. An additional disadvantage is the fact that it is easily taken, has no tell-tale odor, and the patient may substitute it for alcohol. In well controlled selected cases it may be of value.

Paraldehyde has a nasty, vile taste, and one certainly does not want to use it as a punitive measure. It is well to explain to the patient who is not acquainted with this drug what he can expect and why it is given. My nurses are instructed to put the given amount of paraldehyde in about one and one-

half ounces of orange juice and stir this briskly until it gets almost to the patient's lips. The patient is encouraged to take it down at one gulp, followed by a chaser of orange juice.

Adrenal cortex, as recommended by Smith,[7] is frequently used with considerable success in the early treatment of an alcoholic patient. However, if used in adequate dosage, it does involve considerable expense, and expense is sometimes an important item with the alcoholic in the general hospital. The use of ACTH has also been reported[7] to be successful in cases of delirium tremens. However, with adequate early treatment as outlined above, severe delirium tremens is seldom a serious problem.

In addition to dealing with the physical aspects of the alcoholic, we certainly must not forget the psychological problem. Probably the worst thing that can happen to an anxietous alcoholic during the withdrawal phase is to be restrained in bed or locked in a room alone. The insecurity that exists in these individuals really shows up at this time, often in the form of aggressive or psychotic behavior, or even both. The entire treatment program must be oriented toward meeting the patient's needs, and not the least important part of this is establishing the patient's confidence and good rapport with the hospital staff.

Rather than restrain the disturbed alcoholic, or even when restraints are necessary to allay panic, it is desirable to have someone with the patient. The mere presence of another person in whom they have confidence provides a source of protection that does a great deal to allay fear and avoid delusions and hallucinations. In most cases this can be arranged for a few nights by friends, relatives, or occasionally volunteers from Alcoholics Anonymous, usually in relays. It is important that this individual not be an authoritative figure, nor should he be excessively solicitous, but simply another individual who gives the alcoholic a source of confidence. Having the patient near a nurse's station where she can step in frequently will often suffice with disturbed individuals. Although these visits may be brief, they tend to alleviate the anxiety that accumulates with loneliness. The well managed alcoholic can be handled with less disturbance than many postoperative patients.

The alcoholic invariably has a lot of things he needs to talk about, and it is interesting how much he can get "off his chest" in a short period to the physician who is not critical, punitive, or condoning but simply tolerant with the problem, understanding, and willing to help. For the average uncomplicated case, five to seven days of hospitalization will usually suffice, but plans for the next step should be made well in advance of dismissal, and the patient should have slept well several nights without sedation before he is ready to leave the hospital.

Almost every Kansas community has additional resources which can be called upon anticipating dismissal, and probably worthy of first mention is the local Alcoholics Anonymous organization. It is certainly not enough to sober a drunk and turn him over to A.A. as one would pull a wrecked car out of the ditch and send it to a shop, but A.A.'s do have a keen insight into the motivation of the individual and a day to day philosophy, a religion, and a form of group therapy that has made an outstanding reputation for this organization. This group can be called upon relatively early in the course of therapy as most organizations have a few men who have had quite a wide experience in the initial interview.

Occasionally a patient will refuse such a contact, but in my experience refusals have been relatively few, particularly when the introduction is arranged with no betrayal of confidence and still no show of authority. The patient's permission can be solicited with a remark something like this, "Would you mind if we called in a member of Alcoholics Anonymous to talk this matter over with you and see if he has anything to offer that you might want?" The patient will usually give it a try. The physician should by all means see the patient at regular stated intervals following dismissal for symptomatic and supportative therapy, even if no regular medication is given.

The Salvation Army offers valuable help as the next step from dismissal from the general hospital in certain types of cases, although their facilities are not available in many of the smaller communities. However, in some areas they are doing a fine piece of work with their long-term rehabilitation program. This is a service that deserves support and encouragement by physicians as individuals as it is one of our valuable adjunctive therapies.

We have many other social agencies, including the county welfare organization, which may have valuable services to offer in selected cases, and of course, sooner or later a number of alcoholic clinics will become a reality.

Another source that is always worth investigation is the individual's own family. It is always worthwhile to make at least some investigation as to the family relationships and what the family attitudes are toward the patient's behavior. Are they punitive, over-protective, dominating, over-solicitous? Are they putting too many demands on the patient, or are they not offering the patient sufficient responsibility? It is quite difficult for a family to have an alcoholic member without showing considerable shame, hostility, or guilt, and this can well be expressed in a deleterious manner toward the patient. Giving the relatives a chance to unload their feelings often helps to soften this attitude. If the listener will manage to keep quiet and do no probing, the relative will frequently wind up by evaluating the patient's assets and

show a desire toward really helping him rather than punishing him for his past or policing him in the future.

I have found it to be a poor prognostic sign to have a husband identify with A.A. if his wife refuses to attend any of the open meetings or associate with the other wives at any of the A.A. functions. This invariably means that she is ashamed, hurt, hostile, not understanding, or has some other feeling which prevents her from considering "our" problem, and she is not in a position to give the husband help that he will need.

I recall talking to one such wife of an alcoholic shortly after she had been inadvertently thrown into the company of a number of other A.A. wives. Her reaction was one of astonishment. She started off by saying, "Once those women found out that my husband was an alcoholic too, I didn't have a secret in the world. They don't ask you if you have a financial problem, social or sexual problem, or if the children are unhappy or anything—they know you have problems like that. You might as well open up and discuss it and get all the help you can. They stopped treating it as something to be ashamed of long ago."

There are many valid arguments against hospitalizing all alcoholics in psychiatric hospitals. In the first place, we do not have enough psychiatric beds to accommodate them, nor do we have enough psychiatrists and clinical psychologists to carry on their ordinary work and assume responsibility for the 3,800,000 individuals in the United States who have an alcoholic problem.[8] Suppose, however, we did have sufficient personnel and beds. We must consider that the alcoholic has already quite well stigmatized himself in the community by the use of alcohol, directly or indirectly; if we add to that the stigma that is frequently associated with psychiatric hospitalization, we have actually increased the individual's burden.

Very few alcoholics require prolonged hospitalization. As a rule, after a few days or even a few weeks at the most, they can be handled on an outpatient basis . . . this means that when the individual has been hospitalized at some distance from his home, the treatment process that has been set up has to be disrupted and a new one established. This offers an excellent opportunity for a relapse. Another strong argument for treating the patient as near his home as possible is the fact that frequently the family plays an important part in the treatment and they need to participate in therapy plans, attitudes, and rehabilitation.

From the standpoint of the average psychiatric hospital, alcoholics present a particular problem in that their needs are not those of any other particular group in the hospital. If alcoholics are to be treated in any great numbers, they certainly require a special section with facilities for adequate examination, adequate medical care, and a program oriented toward a short period of hospitalization in contradistinction to the relatively longer period required by most other psychiatric cases.

From the standpoint of the alcoholic himself, there are a few things of considerable importance. In the first place, the attendant court procedure and transportation to a psychiatric hospital frequently precipitate more hostility in an individual who is already handling his hostile feelings poorly. Occasionally it engenders a feeling of hopelessness which certainly must be overcome before any real progress can be made. Then, there is the matter of timing. The individual may be receptive at one time, but several days or weeks delay in the admission procedure may result in total loss of an excellent opportunity.

During the early phase of the treatment, there is little that the psychiatrist has to offer that the general practitioner or internist cannot do. Psychiatric evaluation of the underlying psychopathological processes is quite impossible before the patient is well "dried out." It is only proper that the screening be done as near home as possible and with as little disruption of social, family, and economic life as expedient. Those individuals in whom further psychiatric care is necessary can frequently be handled on an outpatient basis or by transfer to the psychiatric hospital when it is definitely seen that that need exists.

CONCLUSIONS

We do have a serious alcoholic problem that the public has shown indications of facing in a realistic and objective manner. Research on this subject has been seriously neglected, and our present facilities are inadequate, but our greatest deficiency at present lies in our failure to organize and utilize the knowledge and facilities we have. It is highly desirable to treat as many alcoholics as possible on a local level, and the general hospital can play a major role in solving this imporant public health problem.

BIBLIOGRAPHY

1. Kansas State Traffic Department. Yearly summary of motor vehicle traffic accidents in Kansas, Jan. 1 to Dec. 31, 1953.
2. Lemere, Frederick: What happens to alcoholics. Am. J. Psychiatry 109:674-5 (March) 1953.
3. Hirsh, Joseph: Alcoholism and the general practitioner, Postgrad. M. 8:5-9 (July) 1950.
Bowman, K. M., and Jellinek, E.M.: Alcohol addiction and its treatment. In Jellinek, E. M. (Ed.): Effects of alcohol on the individual; alcohol addiction and chronic alcoholism, Yale University Press, 1942, pp. 3-80. (discussed in Hirsh's article).
4. Knight, R. P.: The psychodynamics in chronic alcoholism, J. Nerv. & Ment. Dis. 86:538-548 (November) 1937.
5. Davis, C. Nelson, and Robertson, Harold F.: Oxygen in treatment of acute alcoholic intoxication, Quart. J. Studies in Alcohol 10:59-62 (June) 1949.
6. Moeller, K. O.: Death from morphine in persons under the influence of alcohol; 16 cases since 1945. Tidsskrift For Den Norske Laengeforening, Oslo, 73:1-8 (Jan. 1) 1953; Ugeskrift f. Laeger, (Dec. 11) 1952; ab. J.A.M.A. 152:288;352, 1953.
7. Smith, J. J.: Medical approach to problem drinking. Quart. J. Studies on Alcohol. 10:251-257 (September) 1949; Treatment of acute alcoholic states with ACTH and adrenocortical hormones. Ibid. 11:190-198 (June) 1950; Endocrine basis and hormonal therapy of alcoholism. New York S.J.M. 50:1705-6 (July 15) 1950.
8. Facts and Figures About Mental Illness and Other Personality Disturbances. National Association for Mental Health, Inc., 1790 Broadway, New York 19, N. Y. 9 pages.

Treatment of the Alcoholic Patient in Kansas State Mental Institutions

George W. Jackson, M.D.

Topeka, Kansas

Additional attention has been focused in recent years on treatment of the alcoholic patient in state mental hospitals in Kansas. Prior to that time, he was frequently treated in the community as a "common drunk," and little effort was made to try to discover the underlying causes of his drinking or to help him with his problem.

As Kansas' mental hospital program developed and more qualified professional personnel became available to treat patients of all types in state hospitals, treatment programs for citizens of Kansas suffering from this disease were begun. Each of the three state mental hospitals—Larned State Hospital, Osawatomie State Hospital, and Topeka State Hospital—now has a formal program for treatment of the alcoholic patient. A description of each of these programs has been prepared by a physician in each hospital as part of this article.

Co-operation of Alcoholics Anonymous and the recently created Commission on Alcoholism has been a major factor in developing the alcoholic program in state hospitals.

During the calendar year 1953, alcoholic patients represented approximately 9 per cent of admissions to state mental hospitals in Kansas. One hundred twenty-two patients were admitted who were diagnosed as alcoholics. These patients spent a total of 7,545 patient days in the hospitals for an average stay of 61.8 days per patient. Ninety-five of these patients received medical treatment, and 104 were given psychiatric treatment.

Forty-five of these patients came to the hospital on their own referral, 9 were referred by their physicians, and the remaining 68 were referred through probate courts on complaint of relatives, neighbors, police departments, county attorneys, Salvation Army, and others.

Most hospitals recommend that the alcoholic patient come to them on a referral order from the probate court, so that treatment may be planned and carried through to a medical conclusion. The "voluntary patient" may decide to leave the hospital before he has received maximum benefit from the treatment. Under present laws, there is nothing the hospital can do except release him, unless his condition is such as to warrant the filing of a complaint with the probate court at that time.

The principal limitation in treatment of alcoholics in our state mental hospitals at present is the lack of sufficient beds and the general shortage of trained personnel. Of course, this applies to patients suffering from other mental illnesses as well.

Additional research into problems of the alcoholic patient is needed. It is significant to note the variations in treatment and approach to the problem in the three mental hospitals. Perhaps a more universally accepted treatment can be developed through their combined efforts.

A summary of the type of treatment offered at each of the state mental hospitals follows.

Director of Institutions, Kansas State Board of Social Welfare.

Larned State Hospital

Earl F. Morris, M.D., Staff Physician

SOBERING THE ACUTELY INTOXICATED INDIVIDUAL

Treatment at this stage is directed toward correcting dehydration and electrolyte imbalance, vitamin deficiency, protein deficiency, and central nervous system irritability.

We handle these patients without restraint, if at all possible, and start intravenous glucose 5 or 10 per cent in normal saline, 2 liters every 12 hours. We do not add alcohol 5 per cent to this fluid since small amounts of intravenous alcohol seem to aggravate symptoms.

Sedation is secured by paraldehyde, 4 drachms every 4 to 6 hours the first day or two, or phenobarbital, 3 grains by mouth or hypodermically for the first 3 days. This may be supplemented by nembutal in 3 grain doses if needed. Of late, we are favoring the latter program instead of the paraldehyde.

Vitamin deficiency is treated by giving thiamine chloride parenterally for the first week.

We withdraw liquor more rapidly than some and rarely permit any after the second day. The first two

days we give 2 ounces of whiskey every 4 hours while the patient is awake and taper off rather rapidly.

If delirium tremens threatens, we give cortisone in 25 mg. doses every 6 hours.

If, for some reason cortisone is contraindicated, we give 30 per cent alcohol intravenously in 50 cc. to 125 cc. amounts every 4 hours for a day or two.

As soon as food is tolerated, the patient is encouraged to take small amounts of solid food and increase it as tolerated.

TREATING THE CHRONIC ALCOHOLIC

After the patient is sober, the program of treating his or her chronic alcoholism begins. While some benefits can be secured by using the principles of group therapy, it is extremely important at this point to recognize the need for individualizing. A program of handling which is helpful to one may actually drive another patient to drink. We have seen this happen.

Since so many alcoholics have psychoneurotic or personality disorders, we give attention to these basic conditions of which alcoholism is merely a symptom. Occasionally, alcoholism is merely a symptom of increased psychomotor activity, and electroconvulsive therapy directed toward alleviating the mania or hypomania aids greatly.

Psychotherapy is directed toward helping the patient understand himself and his problems; it frequently includes conferences with wife, parents, or close friends. This therapy is handled in groups and individually. Early use is made of Alcoholics Anonymous, and most patients are urged to attend A.A. meetings. We have a group which meets in our hospital library each week and consists entirely of our own patients and employees. There is also a strong group in Larned which meets weekly, and our men attend regularly. We feel that A.A. is one of the most effective measures, if not *the* most effective measure, we employ. However, there is an occasional patient this group does not reach, and such patients should not be over-urged to attend. The program simply does not appeal to an occasional patient, and we must individualize our treatment.

We use antabuse selectively and only if the patient is heartily in favor. If the patient's home and community life is such that taking the first drink is an especial problem, then antabuse is discussed with him. If he decides to take this treatment, we follow the usual procedure of checking him carefully in a physical way. Extensive laboratory work is done, including electrocardiogram, electroencephalogram, basal metabolic rate, blood examination, urine analysis, chest x-ray, and liver function tests, together with the fasting blood sugar. Some of these tests are routine for all patients admitted to the hospital, whether alcoholics or not.

If all tests are favorable, antabuse is given, followed by a test dose of the patient's favorite alcoholic beverage. He is later released from the hospital with instructions for continuing to take antabuse. He also returns to the hospital for rechecks over a period of several months.

We are giving less and less antabuse. The patient must be thoroughly sold on the idea of sobriety if he continues to take it, and if he has that attitude, he probably doesn't need the antabuse.

If there is one point we stress in our psychotherapeutic handling before the patient is sent out, it is the point of insisting that he learn to stand on his own feet, make his own decisions, and be responsible to himself. Too often, these patients come to us having been closely supervised by relatives and friends in such a way that those people are making all major decisions. Liquor bottles are hidden, money is restricted, use of the car is prohibited, and associates are selected. If, under such a program, the alcoholic outwits his supervisors and becomes intoxicated, he regards it more *their* fault than his own because they were not smart enough to hold him in line. If, however, *he* is making the decisions, then he has no one to blame but himself.

After patients have been "dried out," we find this difference in handling is important. As a result, we early give full ground privileges, later town privileges, and soon the patient is running his own life 100 per cent and we are not watching to see if he goes on a "binge." Usually, he decides when he should go home and how. Always he is permitted to make whatever trips he wishes while here. An alcoholic usually thrives on the feeling that he is a responsible person again, capable of making his own decisions and even helping someone else who is down.

We make use of all adjunctive therapies when they seem appropriate. Recreational and occupational therapy are useful in many cases. Our chaplain and our social workers also play an important part in giving the alcoholic a new outlook on life and a new sense of values. Merely being in a state institution where he sees to what levels the human mind can deteriorate if proper habits of living are neglected is in itself a sermon to many, and they often mention this phase of treatment and its benefits.

We seldom keep chronic alcoholics here longer than six weeks. Some of them are here only a month. Hospitalization that is too prolonged is more likely to do harm than good.

It is too early to give accurate figures on results. "Cure" is a relative term. All we can say at present is that possibly something over 60 per cent have gone out and stayed out. They seem to be leading useful lives. However, it will be at least five years before we can release statistics that can be considered accurate.

Osawatomie State Hospital

J. E. C. Morton, M.D., Staff Psychiatrist

The present program for alcoholism has been in operation at this hospital since January 1, 1954. Consequently, we are not yet in a position to report results; however, I think it is fair to say that this has dealt with some of the problems in this difficult area fairly successfully. The basis of this program is as follows.

Recognizing that there is nothing to be gained by a short drying out period and a quick discharge for these patients, we are accepting them on a basis of a 90-day referral by the court. This is the preferred method of admission.

The treatment itself consists of a six weeks course (Table I), at the end of which time the patient is dismissed with the warning that a second referral by the court will entail a stay of a full period of 90 days. Broadly, the course consists of a brief (48-hour) drying out period during which the patient is given sedation as required. This is followed by sedative and tonic hydrotherapy as required, which may extend as long as necessary in the individual case. The rest of the patient's time is taken up by occupational and recreational therapy and working around the wards. The patient is expected to do chores of a necessary kind for other people without expectation of special reward for himself other than the attaining of ground and canteen privileges. These privileges are promptly withdrawn if the patient ceases to cooperate with the program.

At the end of 30 days, the patient's physical health

Routine Orders for the Alcoholic Patient

Six-weeks course

1st Day

1. EEG (if cooperative)
2. Skull X-ray
3. Icterus Index
4. N.P.N.
5. Urinalysis
6. C.B.C. and Nb.
7. Hydrotherapy—continuous tub P.R.N.—2 hours
8. Chloral Hydrate 2 drams (2) every 3 hours for not over 2 doses—if patient has been on antabuse
9. Paraldehyde 2 ozs.—if patient has not been on antabuse
10. EKG
11. Routine Blood Sugar—Glucose tolerance test

2nd Day

1. Chloral Hydrate 2 drams—once for the antabuse patient
2. Paraldehyde 1 oz. if patient has not been on antabuse
3. Hydrotherapy—tubs a. m. and p. m.—1 to 2 hours
4. Needle showers P.R.N.—at least B.I.D.
5. Brewers Yeast *one* Tab. B.I.D. for one week

3rd Day

1. No sedation at all
2. Put to work if able. Or to occupational therapy
3. Needle showers—daily for 1 week
4. Tubs—daily

4th Day

1. Tubs—daily
2. EEG (if not done previously)
3. Liver function tests as requested by physician

5th Day to end of course

O.T., R.T., or employment, with daily tub and needle shower, outdoor exercise.

14th Day

1. Start Antabuse Therapy 4 tablets H.S. (5 grn.)

15th Day

1. Antabuse—Tabs. 3 H.S.

16th Day

1. Antabuse—Tabs. 2 H.S.

17th Day

1. Antabuse—Tab. H.S.

18th Day

1. Antabuse—½ Tab. each night. Continue to 30th day and onwards.

After 30 days of antabuse, give first antabuse trial. Then in one week, another.

Patient leaves at the end of six weeks with a week's supply of antabuse.

is usually greatly improved. Meanwhile he has started on antabuse, and on the 30th day after admission he is given his first antabuse test with alcohol. Medication with antabuse is continued for a week when the second antabuse test is given, and the patient is then discharged with the warning quoted above, a week's supply of antabuse, and instructions to obtain further supplies through his own doctor.

Group psychotherapy is another part of this program which has its importance, but it is not heavily emphasized for good reasons. Among these is the fact that the patient does not stay in the hospital for a long enough period, and little can be expected from group psychotherapy under such conditions.

As soon as the patient comes out of the toxic condition with which he was admitted, it is explained to him somewhat forcibly that he cannot expect miracles to be performed here or anywhere else, either by means of antabuse or any other crutch; that the question of whether or not he remains an alcoholic is for him to decide. We will help him to "straighten out," but we cannot do it for him. This is his problem which he will have to solve from within his own resources. All we can do is to show him how. This is reiterated at intervals throughout his stay in the hospital. Alcoholics are treated exactly the same as other patients in the hospital and are not allowed to consider themselves a class apart or privileged characters.

It is frequently impressed upon them that they are immature, overly dependent characters who need to grow up, do things for other people, and cease to be dependent for their needs. They are also encouraged to attend meetings of Alcoholics Anonymous, either in the town or the group which meets in the hospital itself, or both. Briefly, they are given every opportunity and encouragement to "reform," and ways in which they may do this are pointed out to them. They are also confronted with their own inadequacies as occasion and opportunity offer.

The above is the program as applied to the common, uncomplicated type of alcoholic without major problems other than alcoholism. Where we have reason to suppose that an underlying psychosis is the cause of alcoholism or is a prominent part of the picture with which the patient enters the hospital, the policy is to deal with the alcoholism first and with the psychosis, separately, later.

There is also a routine laboratory investigation which is designed to discover co-existing physical ailments such as diabetes, impairment of liver and kidney function, and organic central nervous system lesions. Chest and skull x-rays are included in this, as are an electroencephalogram and an electrocardiogram. Such physical conditions as are revealed by these testing processes are dealt with to the best of our ability and within the framework of the hospital.

In cases of serious brain damage, other central nervous system lesions, or active psychosis, it is the policy, if this is considered to be in the patient's best interest, to have him committed and allowed to return home so long as he is able to make a successful adjustment. If he proves himself unable to do this, he becomes a custodial case and is transferred from the Receiving Building to the Continued Care service of the hospital.

Modification of this program from time to time as it seems advisable may be made in the light of experience in its operation.

Topeka State Hospital

Stuart Averill, M.D., Psychiatric Resident

The alcoholic program of the Woodsview Section of Topeka State Hospital, still in its infancy, grew out of a spontaneous interest on the part of all members of the section in the alcoholic patient and in the real need for evolving an effective method of meeting his demand for psychiatric treatment.

We felt that the alcoholic patient is a sick person, no more responsible for his activities when drinking than is the schizophrenic for his delusional system or his psychotic actions. Because of his intact intellectual functions and his ability to present himself as an extremely lovable, although mistreated person, the alcoholic attracts individual interest from hospital personnel and the investment of considerable therapeutic enthusiasm. All too often the results of intensive individual efforts at therapy are disillusioning, when the alcoholic begins drinking again, and pessimism may develop about any form of treatment. With these ideas in mind, it was felt that all decisions regarding alcoholic patients should be on the section level with all the staff participating.

Alcoholic patients and their treatment program have been subjects of a two-hour meeting every two weeks for the past eight months. In our approach to the problem of the alcoholic, we have utilized our general knowledge of the treatment of any mental illness and have been non-punitive and non-moralistic, but authoritative. The program and the

individual needs of the patients in it are under constant consideration. This has led to a flexible structure.

In the 8 months that the program has been evolving, 14 patients have been included in the group of alcoholics. Selection of patients for the group has been made by using as broad criteria as possible. The only patients with a presenting symptom of alcoholism who are excluded from the group are those with psychotic reactions or organic brain disease. The apparent conscious motivation of a patient has not been found to be a particularly useful indication of his ability to benefit by a structured treatment program.

On admission, as with all patients, the alcoholic is assigned to one psychiatrist who studies him as a whole person, obtains the story of his life, examines him carefully from a physical and mental standpoint, and determines what assets he has which might be used in his treatment. In addition, the psychiatrist requests psychological tests which give further information about the patient's emotional life. Social service workers meet with the patient's family to obtain information about him and to determine if casework with the family will be of benefit.

At the end of three weeks, the patient is seen in a meeting of the section staff, available information is reviewed and discussed, and a decision is made as to whether or not he should be included in the alcoholic program.

The alcoholics in the program are on a closed ward with other mentally ill patients, most of whom are quiet and undisturbed, although most are psychotic. The group has to abide by the general rules of the hospital and of the closed ward, although all of their activities are undertaken as a group. The patient continues in the structured program for a period of 90 days, at which time he is again seen in a staff conference, and further treatment recommendations are made.

The psychiatrist who initially examines the patient maintains contact but is not actively concerned with his treatment. One ward physician ministers to all patients in the group, takes care of their physical needs, and supervises the structured program. Another psychiatrist directs group therapy meetings.

The daily routine of the group consists of four hours of semi-skilled labor in the mornings under the supervision of an industrial therapist. Work has consisted of pouring concrete, plastering, and performing general repairs about the hospital. In the afternoon, 1½ hours are given to shop and craft work with other mentally ill patients, and it is hoped the alcoholic will select a hobby and learn to enjoy working with his hands. In addition, there are two half-hour periods of music therapy a week especially for this group, where one patient selects a program of music which pleases him and presents it to the rest of the group for their listening enjoyment. The group has one hour each afternoon to go to the canteen and to walk about the grounds. The group attends regular hospital activities in the evenings—dancing and movies.

An effort is made to meet requests of individual patients for activities outside the regular program if this is felt to be therapeutic. There is a one-half hour administrative meeting a week with the ward physician at which time complaints regarding the structure of the program are heard and discussed.

There are two hours of group therapy a week, led by a psychiatrist, in which the section psychologist acts as an observer and recorder. An effort is made at these times to get patients to look at themselves and their problems realistically and to bring home the fact that problems which make them drink are internal problems for which there is no magic solution. An effort is made to convey some idea of the nature and goals of psychiatric treatment. At the same time, the therapist does not lecture or moralize but attempts to bring the group to realize these ideas independently by asking questions and by crystallizing ideas presented by the group. The content and emotional interplay of these therapy meetings is discussed and evaluated in regular meetings of the section staff.

Visitors are permitted on Saturday and Sunday afternoons only. The regular church facilities of the hospital are made available to the group. Through the co-operation of Alcoholics Anonymous, there is an open meeting at the hospital once a week which members of the group are encouraged to attend.

There is a great deal of group cohesiveness, and each member of the group takes considerable interest in the welfare of his fellow members. Most free time is spent in conversation about themselves and their drinking problems. They have contributed to the sociability of the ward with coffee hours and by playing games and talking with other patients. Psychiatric aides on the ward assist in seeing that alcoholics are following the program outlined for them and are sometimes able to point out to the alcoholic that some aspects of his daily behavior are childish and immature.

In essence, this is the initial phase of treatment o. the alcoholic as it has evolved. At the end of 90 days, the patient is again seen and discussed by the staff, and a re-evaluation of his level of adjustment is made. At this time, the recommendations are much individualized, depending partly on the patient's demonstrated motivation, and may include the opportunity for individual psychotherapy with further hospitalization, the freedom to seek a job outside and live in the hospital, trial visits to home and family,

or dismissal as having received maximum benefit from hospitalization.

Although most patients feel considerable urgency to leave the hospital and to try themselves out at this time, it has been our impression that a gradual separation from the hospital's control and support works to the best advantage of the patient.

Those patients who have seemingly benefited the most by the hospital program are those who are most willing to accept the recommendations made at the time of impending release. Alcoholic relapses also have been most common at this critical time. If the patient is experiencing his first admission, he is usually given freedom to accept or reject our recommendations, but if he has already demonstrated an inability to exercise good judgment in planning his future and accepting responsibility for himself during this admission or after a previous hospitalization, we assume a more authoritative attitude and take more responsibility toward seeing that his hospitalization is continued.

We have felt that the hospital's ultimate responsibility is commitment of the patient, if it becomes apparent that he is completely unable to manage his life outside the hospital. We have recommended this in only one case thus far.

It is too early to evaluate our results, but the program to date has attracted the ideas, enthusiasm, and criticism of the staff, who, acting as a group, have tried to maintain a consistent and objective approach to the treatment of the alcoholic patient.

Alcoholics Anonymous

Mr. X

The scene is a hotel coffee shop, during the lunch hour. At a table is seated a group of four men, all of whom appear well groomed, and they seem to radiate a definite sense of well being and general happiness.

If you should ask who they are, the answer would be, "That's Bill J—, president of a large insurance company; next to him is John M—, one of our town's most highly regarded physicians; the third is George D—, the prominent real estate man, and last is Richard G—, a splendid attorney who is active in our town's civic affairs."

Certainly that is an obvious and seemingly comprehensive answer. Yet there is a vitally interesting matter which binds those four men together as close friends. The fact is they are all *recovered alcoholics*. If you were to talk with each separately, you would find that they could make practically identical statements, "Alcoholics Anonymous helped me to save my life."

The Alcoholics Anonymous organization has been active in Kansas for a little more than a decade. The first small group began in Kansas City and from there spread across the state. Currently there are about 60 active groups throughout Kansas with more than 1,000 active members.

The organization is unique in that it finances itself by voluntary contributions from members and that it is free from a maze of hindering rules and regulations. The qualification for membership is basic, simply an admission by men or women that alcohol has made their lives unmanageable and that they, as individuals, sincerely and honestly want to free themselves from the chains of alcohol.

Many people are curious as to the "why" of the anonymous portion of the organization's name. It is largely because in anonymity lies one of the greatest sources of strength to perform the work. It is a protection to the family of the new member. It is an equal protection to the person who becomes a member. He knows that his name will not be used and that he will not be quoted without his direct permission.

The whole creed of A.A. is compactly revealed and contained in the organization's "Twelve Steps" to recovery. They are:

Step One: We admitted we were powerless over alcohol . . that our lives had become unmanageable.

Step Two: Came to believe that a Power greater than ourselves could restore us to sanity.

Step Three: Made a decision to turn our will and our lives over to the care of God as we understood Him.

Step Four: Made a searching and fearless moral inventory of ourselves.

Step Five: Admitted to God, to ourselves and to another human being the exact nature of our wrongs.

Step Six: Were entirely ready to have God remove all these defects of character.

Step Seven: Humbly asked Him to remove our shortcomings.

Step Eight: Made a list of all persons we had

harmed and became willing to make amends to them all.

Step Nine: Made direct amends to such people whenever possible, except when to do so would injure them or others.

Step Ten: Continued to take personal inventory and when we were wrong, promptly admitted it.

Step Eleven: Sought through prayer and meditation to improve our conscious contact with God as we understood Him, praying only for knowledge of His will for us and the power to carry that out.

Step Twelve: Having had a spiritual awakening as the result of these steps, we tried to carry this message to alcoholics and to practice these principles in all our affairs.

The last step is known as the "work" step, and A.A.'s are happy in the knowledge that they are able to help someone else recover—and to do it without thought of public recognition or personal, selfish gain.

This creed has been described as "Christianity in Shirtsleeves" or "A Practical Philosophy for a Way of Life that Works." Physicians across the nation whose work has brought them into contact with A.A. people and the A.A. organization have said, "A.A. is a modern miracle! It is one of the most inspiring activities of our time."

Thousands of men and women throughout America have been restored to their families and their work. Fully authenticated case reports tell of innumerable men and women in all walks of life who, after their affiliation with A.A., have created brilliant, worthwhile careers in both professional and personal life.

One of the basic tenets of A.A. is "Take it easy." The member is instructed to live one day at a time. In other words, a man simply says, "I'm not drinking today." As the days accumulate, the sobriety record becomes months, and eventually it is charted in years. Many members have 10 years and more to their credit and bear eloquent testimony to the soundness of the A.A. plan.

As men and women mature in A.A. affiliations, they are quick to realize and praise the work of other sources in battling the dread disease of alcoholism. A.A. members are not narrow or bigoted, and they observe and encourage the work of such bodies as the Yale Clinic on Alcohol Studies and other medical and professional groups. They know and understand the essential value of a sound body as an attribute of sound being.

It has been aptly remarked that the A.A. philosophy could be adapted to any person's life as a safe, sane, and workable method of bringing peace of mind, physical well being, and complete harmony of spirit, mind, and body. The A.A. prayer says it all, "God grant me the serenity to accept the things I cannot change, courage to change the things I can, and wisdom to know the difference."

If we are to build and maintain the strength required to cope with the problems of this age, we must cooperate one with the other, every section with all others, each group with its neighbors. This means domestic unity. . . . Unity does not imply rigid conformity to every doctrine or position of a particular political figure. But it does require a common devotion to the cardinal principles of our free system, shared knowledge and understanding of our own capacities and opportunities and a common determination to cooperate unreservedly in striving toward our truly important goals. This type of unity is the true source of our great energy—our spiritual, intellectual, material and creative energy.

—Dwight D. Eisenhower

Sodium Pentothal Solution, an Adjuvant in the Treatment of Acute Alcoholism

DeMerle E. Eckart, M.D.

Hutchinson, Kansas

The problem of alcohol, with its slowing effect on conduction speed in the central nervous system, is unique with the nature of our particular cultural demands. The high efficiency that is the backbone of our mass production techniques requires maximum speed of conduction from the organs of hearing and seeing to the cortical association centers via the main common pathways to the neuromuscular end organ. Since this is not an outstanding trait of other cultures, there is marked variation of classification and definition, ours being more rigid and having greater moral implications.

The alcoholic has been a frustrating, cumbersome, and irritating problem to the physician, nursing staff, and hospital. The alcoholic patient is a poor economic risk and requires close supervision. Attempts at jumping out windows or self-injury are not uncommon. If, occasionally, a successful method of physical restraint is found, a noise problem results that is disturbing to other patients. I have been impressed in the past by the poor response of the patient to drug therapy, especially during the evening hours. The hormone drugs are expensive and have not answered this problem.

In our particular area, one type of so-called problem drinker is the man who stays dry for a period of two to six months, goes on a "bender" for one to three weeks, and then contacts the A.A. or his friends, seeking, through his family physician, some way of drying out. There is also the middle-aged man who has gradually increased his intake over the last 12 to 24 months and suddenly finds himself unable to keep his job or business going. These men generally seek out their family doctor for help. Last is a more discouraging type; I have seen the "skid-row" type of drinker on two separate occasions.

Sodium pentothal has been widely used as an anesthetic agent for minor and major surgical procedures, alone and in combination with other agents. In later years, it has been used as an agent in psychotherapy, and Dr. Voegtlin used a .2 per cent solution with 5 per cent glucose in 500 cc. amounts intravenously, following aversion treatment in conjunction with psychotherapy.

The procedure was first used on a 44-year-old male, who, on the third post-alcoholic day, developed an acute toxic psychosis, broke a water glass, and cut a medium-sized vein in his neck. Following surgical repair, he was kept under control with sodium pentothal solution until he recovered. This was approximately five days.

The following is the procedure used for treatment of acute alcoholism on 75 hospital admissions in the Grace and St. Elizabeth Hospitals. If the patient is combative or resistive, or if there is a suspicion that the patient has had a large intake of alcohol just before starting to the hospital, he is put to sleep with 10 to 15 cc. of 2½ per cent sodium pentothal. In the latter case, the stomach is thoroughly emptied. The patient is started on 1000 cc. of 10 per cent glucose and water or saline containing 2 gm. sodium pentothal intravenously, using a size 21 needle, thus giving adequate opening in the vein to vary the speed of intake between 10 and 80 gtt. per minute. Intake is about 80 gtt. per minute for the first five minutes. The rate is slowed to between 20 and 40 gtt. per minute when the patient begins to get drowsy, with the nurse checking the patient at close intervals for the first half hour.

Insulin, in small dosage of 10 to 15 units, may be given on admission. I generally wait until morning, stopping the sodium pentothal approximately two and one-half to three hours before breakfast and giving between 15 and 50 units of regular insulin one hour prior to breakfast and sweetened fruit juices. Atropine sulfate is given to control increased secretions that appear in some patients.

Insulin and glucose, in many cases with added vitamins, have been widely used for many years to hasten recovery of these patients. Noyes recommends 40 to 60 units on admission to produce relaxation and sleep, followed by a large glass of sugar and fruit juice and a large meal in two and one-half hours. The reported beneficial effect does not appear to be solely due to increased rate of removal of alcohol from the blood. Smith and co-workers found no significant change in the rate of removal of alcohol from the blood with various combinations of glucose, vitamins, and insulin.

I have not been able to mix vitamins with the .2 per cent sodium pentothal solution, and I do not use

Although this paper was not submitted through the Kansas Commission on Alcoholism, it is included in this special issue of the JOURNAL since it relates to the subject covered by the commission in other papers in this section.

them unless there has been a fairly long period without food. Figueroa, in his study, found need for vitamins in only .06 per cent of cases.

During the first three days, with the exception of the day of admission, the .2 per cent sodium pentothal solution should be given only during the night hours, starting between 7:30 and 11:30 p.m. If delirium tremens or toxic psychosis develops, it will generally be between the third and fifth days; at this time the solution may be used continuously until the patient is able to control withdrawal symptoms. The longest period of time, with the latter, has been five days; however, it seldom lasts more than three days. A retention catheter is always inserted under these circumstances.

Hypersecretions occluding the airway create a problem with some of the patients, particularly those whose intake just prior to admission was heavy. Atropine sulfate gr. 1/75, if given on admission, is helpful in preventing this complication. The suction machine and Guedel airway are kept handy until the atropine has had time to take effect. The Guedel airway works well on the patient with dentures.

Restraint requirements are to keep the patient from rolling out of bed. Bed rails are routinely used while the patient is asleep.

It is my opinion that alcohol first suppresses the medullary portion of the adrenal gland with a resultant slowing of conduction time of the main common pathways of the central nervous system with more associated tracts at the thalamus being used, resulting in more depth of emotion or "organ feeling" to come to play in human relationship. Second, when the alcoholic stops eating, alcohol supplements glucose in the adrenal cortex with a suppressing effect on the cortical functions of the adrenals. This is when the acute alcoholic seeks relief because of resulting depression, incoordination, nausea, and often vomiting, diarrhea, and abdominal pain.

It is my opinion that insulin and glucose therapeutically suppress medullary over-sections and stimulate the functions of the cortex of the adrenals. This accounts for symptomatic improvement resulting from these two chemical agents and explains how they can attain the same results as hormone drugs. Sodium pentothal takes over the immediate job of suppressing the medullary portion of the adrenal glands, allowing time for the slower effect of insulin and glucose.

I have discontinued the use of hormone drugs routinely. I have used the hormonal drugs two times with the two chronic "skid-row" alcoholics who, on the third day following admission, developed a clinical picture resembling the Waterhouse-Friderichsen syndrome. These two patients had a dramatic response to large doses of ACTH and penicillin.

CONCLUSIONS

The sodium pentothal-glucose solution has enabled our general hospitals to handle the most difficult alcoholic with the usual physical facilities and nursing methods present in all hospitals.

The nurse and physician are using chemical agents with which they are familiar, without the requirement of specialized training or excessive risk.

The risks are respiratory depression, infection, and severe insulin reaction. The first two are common problems with alcoholics. The third is not an uncommon problem with any physician treating diabetes. None of these have been serious, and all have been handled by the usual methods without difficulty.

SUMMARY

I have outlined a procedure using sodium pentothal-glucose solution intravenously in conjunction with subcutaneous insulin. I believe this simplifies the sedation and nursing problem created by the acute alcoholic. I have given my opinion of the action of alcohol on the adrenals and the central nervous system and the underlying therapeutic mechanism of this combination.

BIBLIOGRAPHY

Collected papers of Shadel Sanitarium 1:59-149, 1948.
Smith, H. W.; McBroom, G. L., and Bell, R. G.: Canad. M.A.J. 63:250-255.
Wilson, Ruth: Mental Hygiene 25:260-290 (April) 1951.
Tintera, J. W., and Lovell, H.W.: Geriatrics 4:274-280 (Sept.-Oct.) 1949.
Prout: Alcoholism, Chronic, Treatment, Med. Clin. N. Am. 595-647 (March) 1950.
Liebout: Alcoholism, Nature and Treatment, Med. Clin. N. Am. 687-693 (May) 1948.
Figueroa, W. G.: Lack of autominosis among alcoholic addicts, J. Clin. Nutr. 1:179-199 (March) 1953.
Cleghorn, R. A.: Adrenal-cortical insufficiency: psychological and neurological observations, Canad. M.A.J. 65 (Nov.) 1951.
Adrenals and some of their relationships, Pfizer Spectrum J.A.M.A. 154:15, 28-29 (April 10) 1954.
Noyes: Modern Clinical Psychiatry ed. 4, 187-188.

The United States, as a first colony in modern history to win independence for itself, instinctively shares the aspirations for liberty of all dependent and colonial peoples. We want to help and not hinder the spread of liberty. . . . That is the spirit which animates us. And if we remain true to that spirit, we can face the future with confidence, knowing that we shall be in harmony with those moral forces which ultimately will prevail.—*John Foster Dulles*

THE MONTH IN WASHINGTON

Editor's Note. The following summary of Washington news was prepared by the Washington office of the A.M.A. for distribution to state and regional medical journals.

During the next three years the federal government expects to help finance the construction of thousands of new medical and dental facilities—diagnostic-treatment clinics, vocational rehabilitation centers, nursing homes, and chronic disease hospitals. Only three strings are attached: the facilities must be non-profit, they must be under medical or dental supervision, and local communities must raise part of the cost.

Legislation establishing the new program was enacted just as Congress plunged into its adjournment rush, and before it had come to final decisions on reinsurance and other major controversial bills in the health field.

The new operation was authorized by amending the Hill-Burton Act (passed in 1946 to assist hospitals) to permit grants to units that do not qualify as hospitals. Under the original Hill-Burton law, grants could be made to rehabilitation centers and diagnostic-treatment clinics only if they were attached to hospitals. Grants could also be made to chronic disease hospitals. The new law authorizes help to centers and clinics operating on their own, a provision Public Health Service expects to be of particular assistance to smaller communities. It also offers aid to nursing homes, which previously were not covered.

In the case of chronic disease hospitals, it is explained that the law offers two new inducements for construction: (1) Money is allocated to the state and earmarked for this particular type of hospital. (2) The federal government will be able to pay 50 per cent or more in all cases, whereas under the old law the U. S. share was as low as one-third in some of the higher-income states.

Grants to clinics, centers, and nursing homes will have to wait on state surveys to determine priorities, according to U. S. hospital officials. However, if local sponsors take the initiative, grants can be processed immediately for chronic disease hospitals, as earlier Hill-Burton surveys have established their priorities. Failure of communities to construct chronic disease hospitals was one of the disappointments of the first Hill-Burton program.

The first year's appropriation will be $37.4 million, increasing over the next three years until the total authorization of $182 million has been reached. The new projects in no way interfere with the regular Hill-Burton grants for construction of hospitals, for which $75 million is available this year.

The final flurry over the reinsurance bill was preceded by a concerted drive by the administration. The President himself interceded with insurance company officials, and Secretary Hobby agreed to amendments in an effort to satisfy the state insurance commissioners. The commissioners, who would have an important role in administering the reinsurance program, at first had flatly opposed it. President Walter B. Martin and other A.M.A. officials were called in for a discussion of reinsurance at the Department of Health, Education, and Welfare, and later Sherman Adams, assistant to the President, also invited Dr. Martin to a White House meeting on the same subject.

As expected, bills for a new program of medical care of military dependents were left stranded when adjournment time approached. Before he introduced his bill on the subject, Chairman Dewey Short of the House Armed Services Committee insisted that Defense Department estimate first year's additional cost of the program. The estimate was $67 million.

The military scholarships bill met the same fate—too much time taken up in drafting a version that would satisfy all executive departments. Under this plan the Defense Department would grant tuition-and-maintenance scholarships to medical and dental students, in exchange for pledges to spend one year in military service for every subsidized year of training. Both bills are certain to reappear next session.

For the current fiscal year, the Department of Health, Education, and Welfare has available $1,663,413,761. The appropriation bill is $10,904,500 more than the administration requested but under last year's budget of $1,927,432,261 (the decline explained by decreased public assistance grants to states). Public Health Service has $228,060,000 for its regular programs.

Pharmaceutical House Honored by A.M.A.

For "pioneering use of television in bettering the health of the nation," the A.M.A. at its San Francisco convention awarded a special citation to the Philadelphia pharmaceutical house, Smith, Kline and French Laboratories.

During the A.M.A. meeting a week-long program of color telecasts was produced and sponsored by the firm for the attending physicians. It was the 58th program the laboratory had presented at medical meetings during the past five years. In addition, Smith, Kline and French had presented "March of Medicine" telecasts for lay audiences since 1952.

PRESIDENT'S PAGE

DEAR DOCTOR:

By the time you receive this letter we will know how well or how badly we have done in our efforts at the state primaries, and should be able to orient a policy for the November elections.

There has been some lack of cohesion, for which I blame myself, but from the reports I get we are doing a pretty good job as a group.

The new committees are becoming more active with their meetings this month, and I expect more after the first of September.

I would like the councilors to note that I expect to call a Sunday meeting on the 22nd or 29th of August and suggest that you contact your councilor if you have any problem that should be brought up at that time.

Perhaps if we survive another month of Kansas weather we can get down to business.

Yours,

Murray C. Eddy, M.D.

EDITORIAL COMMENT

Approach to Problems of Alcoholism

Today alcoholism is considered as a valid health problem, an illness, a disease. Increased recognition is being given to the fact that alcoholics are sick individuals, worthy of help, and that they can be helped. For that reason, the Kansas State Commission on Alcoholism welcomed the opportunity to provide material for a special issue of the Journal of The Kansas Medical Society devoted to problems of alcoholism.

The commission realizes that the family doctor is ordinarily the first individual the alcoholic contacts for help. Proper attitudes and approaches are invaluable to the medical physician when the alcoholic comes to him. With a study of the problem, a sincere interest in helping the alcoholic get well, and the innate and all important belief that the alcoholic is a valid patient worth helping, physicians can expect to meet with increasing success in treatment.

The offices of the Kansas State Commission on Alcoholism were opened in the Medical Society Building in Topeka last October. The commission, established by the 1953 session of the Kansas legislature, is composed of five members. Members, who serve without remuneration, are appointed by the governor for four-year terms. By law the commission is composed of two physicians, two alcoholics who have been recovered for two or more years, and one attorney. Present membership of the commission includes Dr. Karl E. Voldeng, Wellington, chairman; Carl E. Ziegler, Coffeyville, vice-chairman; Dr. Z. Miles Nason, Kansas City; Allen O. Hendricks, Wichita, and Dr. George W. Jackson, Topeka. Lewis W. Andrews was employed by the commission as executive director.

The functions of the commission include:

1. Study of the problem of alcoholism in Kansas, including not only the number of cases but methods and facilities available for the care, custody, detention, treatment, employment, and rehabilitation of alcoholics.
2. Education on the illness of alcoholism.
3. Treatment and rehabilitation of alcoholics.
4. Research into factors leading to alcoholism.

The commission is neither "wet" nor "dry." The only concern is to attack the problem of alcoholism, to help the alcoholic recover and, through education and rehabilitation, make a realistic step toward *prevention.*

Reliable studies indicate that approximately 65 per cent of the population in the United States over 15 years of age use alcoholic beverages. Of this 65 per cent, approximately 6 per cent are alcoholics. These studies further indicate that a vast majority of alcoholics are in the age bracket of 35 to 55, and the ratio of male to female is about 6 to 1. Alcoholism is no respecter of social or economic status.

The commission recognizes alcoholism as a medical problem. They earnestly solicit comments and suggestions from members of the medical profession in regard to their program.

—*Lewis W. Andrews*

Federal Health Programs

PART II

The second of a brief series of articles on federal health expenditures considers the Veterans Administration, the Department of Defense, and the Department of State. The first article discussed the Department of Health, Education, and Welfare.

The Veterans Administration, during the fiscal year just concluded, spent, according to a report from the A.M.A. Washington office, $747,415,264 on health. This was budgeted through 10 major services.

Administration used $7,757,900 to pay salaries, travel, and other expenses for maintaining the central office and six area medical offices in the United States.

VA research received $5,500,000, mostly for projects conducted within Veterans Administration hospitals. By way of example, $1,527,600 is allotted for atomic medicine, $800,000 for testing prosthetics, $2,500,000 for research in general medicine and surgery, $150,000 for tuberculosis research, $125,000 for neuropsychiatric research, and smaller amounts for certain other programs.

Construction projects cost $38,685,664, of which just over half is allotted for existing hospital construction and the remainder is earmarked for new buildings, plans, etc. Significant among the latter are two proposed neuropsychiatric hospitals, each for 1,000 beds, to be located at San Francisco and at Topeka.

Supply depot operations will order, store, and disburse $1,350,000 worth of medical supplies to the various hospitals and other locations for use by the Department of Medicine and Surgery.

Contract hospitalization last year cost $20,583,100. This represents payment to hospitals for veterans in other than VA institutions and figures at an average daily census of 5,831 patients. According to the report, the largest single entry is for psychotic patients with a total of 3,084.

Domiciliary care cost $24,248,200. The VA operates 17 domiciliary institutions, of which all but three are connected with VA hospitals to care for

unemployable veterans who do not need full hospital care.

The out-patient care program cost $92,677,900. Two-thirds of the total was spent in clinics operated by the VA. About $7 million was paid to physicians and $23 million to dentists under home-town care contracts.

By far the largest cost grows out of the VA hospital program which amounted to $555,000,000 during the past fiscal year. The VA currently operates 163 hospitals with a total of 114,000 beds. As of several months ago they claimed to be operating at 90 per cent of capacity or with a daily occupancy of about 102,000 patients. Among other statistics only remotely related to this subject are the following: that of the total, 35.6 per cent were service connected, 63.8 per cent were non-service connected, and .6 per cent were non-veteran cases.

Also not directly related to this subject but of possible interest is some information obtained from the A.M.A. *Fact Book on VA Medical Care*. This has been roundly criticized and undoubtedly is out of date at the present time. However, it appears that in 1924 there were 5 million veterans in the United States; in 1953 there were 20 million with this figure increasing at the rate of 1 million per year. In 1924, 16 per cent of the adult male population were veterans; in 1953 it was 40 per cent. Again, if the statistics are reliable, the average length of stay per patient in the field of medicine and surgery within a veterans' hospital is 30.4 days. Under similar circumstances in other general hospitals the average length of stay is 7.5 days.

The two remaining divisions of medical expense under the direction of the VA are medical education and training, which cost $1,300,000, and expansion of 100 VA clinics, most of which are located in regional offices, which cost $312,500.

The Department of Defense operates four services at a total cost of $533,311,000. Almost half of this is spent by the medical department of the Army, including salaries for military personnel, including doctors, but not including the operation expenses of 58 Army hospitals.

The Navy spent $161,429,000, of which 78 million was for salaries, and, like the Army, this total does not include maintenance and operation of certain hospitals. The Air Force Medical Services spent approximately $132,801,000, of which 87 million is for salaries. The final category is $87,000 for the assistant secretary of defense.

The Department of State operates three distinct medical programs under a total budget of $14,127,733. The Department of State operates the World Health Organization, of which the United States contribution is one-third of the total W.H.O. budget. Those interested in this subject may wish to learn that administration and salaries consume 41 per cent of the budget, consultation to foreign governments 35.6 per cent, field programs 13.6 per cent, organization meetings, 3.8 per cent, fact gathering and sanitary services 3.8 per cent, and expert committees studying various diseases 2.2 per cent. As stated above, those percentages represent the total expenditure, of which the United States pays one-third.

The second State Department health project is the Pan American Sanitary Bureau with a budget of $1,320,000. This is affiliated with the World Health Organization but operates under a separate accounting system. Here the United States contributes 66 per cent of the total cost. Items such as field personnel training, insect control, venereal disease control, and environmental sanitation represent the larger expenditures.

The final item of the largest of the three is the International Children's Emergency Fund with a budget of $9,814,333. The above figure is the United States share or 60 per cent of the total operating expenses of a program that provides milk, food, and other services for children in disaster areas.

The above account again has been an attempt to analyze federal health expenditures as budgeted under three of the 19 separate departments which combine to participate in more than 60 health programs. A third article on this subject will continue the discussion to describe the program of the Department of Labor, the Department of the Interior, and others.

Vaccine Evaluation Phase

Editor's Note. The following editorial, prepared by the National Foundation for Infantile Paralysis, Inc., is published at the request of Dr. Hart E. Van Riper, medical director of the foundation. It is of particular importance here since Kansas is one of the test areas in the polio vaccine trial.

More than 600,000 children have completed three inoculations, in the field test of the trial polio vaccine developed by Dr. Jonas E. Salk of the University of Pittsburgh. The emphasis now shifts to the evaluation study under the direction of Dr. Thomas Francis, Jr., University of Michigan School of Public Health. The validity of the evaluation is dependent upon data gathered on poliomyelitis cases in the test groups, *including those children in the first three grades who did not get vaccine.*

In addition, data on cases among family members of participating children are an integral part of the study. Since the number of poliomyelitis cases among the test groups may not be large, it is essential that all cases are completely reported. Early diagnosis, prompt reporting and follow-up, and the securing of *necessary epidemiological information and laboratory*

specimens are important factors in the evaluation.

An outline of procedures and copies of necessary forms have been sent to local and state health authorities. It is important that physicians in areas where vaccinations were not given cooperate in the study by notifying local or state health officers of cases occuring among children who participated in the trials and then migrated to another area and children who go to summer camps. Local health officials also need information on participating children who receive injections of gamma globulin.

This phase of the study will depend, to a large degree, on the wholehearted cooperation of practicing physicians.

For the specific determination of the alcohol content of the blood, chemical methods remain the most satisfactory. One method (Widmark's) in particular can be applied to very small blood samples and the results, checked in hundreds of thousands of tests, may be considered as reliable and accurate, even in the case of persons suffering from diabetes.

The estimation of alcohol in the breath gives reliable but less accurate results and should be used only when blood assay cannot be carried out.—*Pan American Sanitary Bureau, World Health Organization.*

Film on Alcoholism Available

A film on alcoholism, case studies of three types of alcoholics tracing the development of the disorder from origin, has been added to the A.M.A. library of motion pictures. Entitled "Alcoholism," this film shows how the roots of the illness are imbedded in personality difficulties often relating back to the formative years of the victim's childhood. It may be obtained from the Committee on Medical Motion Pictures, A.M.A., for a service charge of two dollars.

Members of the Kansas Medical Society should notify the JOURNAL of any change of address.

SERVICE SEPARATIONS

As a service to physicians and communities in this state desiring additional medical personnel, the Journal of the Kansas Medical Society will publish in this column each month the names of medical officers who will shortly be separated from the armed forces. These are men who volunteered from Kansas, and many of them will probably be interested in finding locations in this state. Anyone interested in contacting these physicians may write to the address here given.

John S. Anderson, M.D.
814 Greenway Court
Wichita, Kansas

Robert S. Brown, M.D.
5009 51st Terrace
Mission, Kansas

George G. Browning, M.D.
316 South 8th Street
Humboldt, Kansas

Dean C. Chaffee, M.D.
417 N. W. Third
Abilene, Kansas

Charles A. Crockett, M.D.
5437 Neosho
Kansas City, Kansas

Jack A. Dunagin, M.D.
1266 Collins
Topeka, Kansas

Raymond G. Elliott, M.D.
6136 Reinhardt Drive
Mission, Kansas

Edgar F. Ewen, M.D.
1022 East 5th
Cherryvale, Kansas

Henry S. Haerle, M.D.
Marysville, Kansas

Wayne E. Hird, M.D.
102 North Byron Road
Wichita, Kansas

Everett E. Howard, M.D.
1708 North Grand
Pittsburg, Kansas

Robert A. Jelinek, M.D.
506 Chestnut Street
Leavenworth, Kansas

J. D. Kabler, M.D.
1506 Parker Avenue
Wichita, Kansas

Alexander H. Milne, M.D.
2420 West 25th Street
Topeka, Kansas

Earl G. Padfield, Jr., M.D.
4412 West 70th
Prairie Village, Kansas

Owen C. Peck, M.D.
2000 Oakland
Kansas City, Kansas

Perry D. Petterson, M.D.
3717 East Murdock
Wichita, Kansas

Ralph B. Reauwe, M.D.
524 West 7th
Junction City, Kansas

Julian W. Reed, M.D.
703 North Pine
Beloit, Kansas

Robert L. Satterlee, M.D.
Macksville, Kansas

Robert N. Shears, M.D.
15 Carlton Road
Hutchinson, Kansas

Grant M. Stevens, M.D.
305 Crescent Boulevard
Hutchinson, Kansas

Charles E. Wilder, M.D.
Winter VA Hospital
Topeka, Kansas

Robert E. Young, M.D.
2400 North 38th Street
Kansas City, Kansas

The Management of Incurable Cancer

Tumor Conference

Edited by Chauncey G. Bly, M.D., and Irwin Joffe, M.D.

Dr. Helwig: Today we are having an important symposium on the management of incurable cancer. It is hardly necessary to emphasize the bare fact that, except for the easily and early recognized epitheliomas of the skin and of some easily accessible mucous membranes, cancer is incurable in a high percentage of cases, and, as such, presents special patient problems.

Dr. Friesen, as a surgeon in collaboration with Dr. Kelly,[1] you published an article in 1950 on the controversial and difficult subject "What Should You Tell the Cancer Patient." Would you lead off today's discussion with some of your very good points on that subject and the other phases of incurable malignancy?

Dr. Friesen: There is no question about the fact that the number of patients who have incurable cancer is increasing. Correspondingly, the responsibility of the physician to such patients has grown to such magnitude that many of us are finding it increasingly difficult to know how best to deal with these individuals.

Dr. Charles Cameron, medical and scientific director of the American Cancer Society, recently estimated that over 220,000 people are or will be dying of cancer in the United States this year; moreover, there are at any one time in the United States about 32,000 people who are terminal cancer patients requiring considerable amounts of medical care. If one were to relate these figures to a community of 10,000 people, there would be 16 patients dying of cancer in a year in such a population.

There is no question that more and more people are now being cured of cancer each year than were cured previously. Yet, as Dr. Helwig has mentioned, the great majority of cancer patients are still not being cured. These latter unfortunates are usually incurable at the time the diagnosis is made. Whether he gives definitive treatment or not, the physician is still the one who must assume personal responsibility for the intelligent and sympathetic care of these patients with incurable cancer.

The first obligation which the physician has to this type of patient is to take nothing for granted. He must be sure that the diagnosis is correct and that the patient has a carcinoma which, according to all the best available information, is incurable. This usually means that one or more biopsies of his patient's lesion will have been examined by a pathologist for a definite diagnosis before treatment with surgery, radiation, or chemicals.

He must also take nothing for granted when he is considering the length of time his patient may live with incurable cancer. It is dangerous to make glib predictions as to how long the patient with incurable cancer will live. Although a physician can make use of statistical probabilities and past experience to predict average survivals for a group of patients with a certain tumor and stage of progression, there are far too many variables for him to confidently predict the course in any individual patient. Too many of us have seen the unhappy situation where a particular patient has lived a much longer or a much shorter time than was predicted. Blind or hasty predictions must be avoided at all costs. Even a conservative and carefully prepared prediction may prove shocking to the patient or relatives, whether or not the prediction turns out to be correct.

If you are sent a new patient who is said to have an incurable cancer, it is your obligation as a physician to review critically all the findings in this patient, repeat the history and the physical examination, and check the previous biopsy report or do another biopsy, so that if this patient becomes your responsibility you personally will know his correct status.

The physician who properly accepts his responsibility should not only have a genuine personal interest in his patient, but he should also carry out active palliative treatment. Success in this area depends as much on the doctor's attitude toward his patient as on the doctor's skill. Any patient who feels that his physician is not personally interested in him, or who is not receiving active treatment of any kind from his physician, is entitled to be dissatisfied. He may go to another doctor, or he may actually be forced by default into the hands of charlatans and quacks, who characteristically give these patients hope that is not justified. The charlatan, moreover, will eventually defraud the patient of his money, economic security, and whatever he can get.

If I were a patient with incurable cancer, I would want my physician to take such an active personal interest in me that I would never feel abandoned or that all hope was gone. He would give me any active palliative treatment that he thought justified, con-

Cancer teaching activities at the University of Kansas Medical Center are aided by grants from the National Cancer Institute and the Kansas Division of the American Cancer Society.

Dr. Bly is a scholar in cancer research of the American Cancer Society. Dr. Joffe is a trainee of the National Cancer Institute.

sidering the stage of my disease and also my financial and mental resources. I would want to be in comfortable, sympathetic home or hospital surroundings. I would not want to be placed in a custodial institution for cursory care, where I might feel that I was just being endured, or even exhibited, as a helpless and hopeless patient, to be occasionally visited for appearances' sake. I would want the doctors, the nurses, the business manager, and everyone else taking part in my care to be genuinely interested in me as a living though struggling fellow human being. I would want each new symptom which I developed to be treated actively as a challenge, thoughtfully and logically, even if only symptomatically. I would not want to be blandly given increasing doses of morphine or other narcotics, for the resultant progressive mental deterioration might well prove to be intolerable.

We, as busy physicians, must still keep genuinely interested in the patient, both psychologically and medically. The physician, the family, and the patient should all be actively engaged in a continuous struggle to maintain the best physical and mental status possible until the overwhelming onslaught of the disease finally kills the patient. The physician must carefully and continuously evaluate the strain and drain on the family.

To maintain the active physical health and the personal integrity of the patient as long as possible, even until death, I personally believe that, in most cases, everyone involved should know the true situation. Too often the confidence that the patient has in the doctor is violated when the truth is withheld or when there is complete evasion of the diagnosis or prognosis. The patient expects the truth from his doctor. The doctor-patient relationship is one which ideally involves trust, confidence, and faith in each other.

We found out in our previous study[1] that the patient usually *knows* what the diagnosis and ultimate outcome are anyway, by the time he has been in the hospital, has been under the care of several physicians, has had x-ray therapy or surgery, and has had his symptoms return again. He usually knows by that time that he has cancer, even though no one has told him. In our study, a high percentage of patients not only knew this but actually entered into the deception. Since neither the doctor nor the family would tell the diagnosis and expectation, they reasoned, "Maybe it's best that I don't know, so I am not going to tell anybody that I do know the diagnosis." Occasionally patients know the situation but subconsciously refuse to believe it and will not ask because of the fear of having confirmed for them what they already know. These practices of group-deception or self-deception are destructive, for fear of the uncertain is worse than fear of the certain. Moreover, the patient may develop great insecurity if both family and physician avoid telling him the truth which he feels he has a right to learn.

Now when I say that a patient should be told that he or she has cancer, I must emphasize that there is a proper time and a proper manner for telling each particular patient. I don't think the physician need tell the patient everything at the time he first makes the diagnosis, or at the time he has operated on him, perhaps unsuccessfully, or while the patient still feels pretty well. Determining when and how to tell the patient is a matter of judgment. We, as physicians, must evaluate our patient to know when we may best tell him that he has cancer and is expected to die of the disease. We must also give him hope, by our personal interest and active treatment, so that he knows that he will live as comfortably and long as possible.

There is no question about whether or not the patient with early cancer should be told. That patient can very possibly be cured. In our hospital rounds we see an increasing number of treated patients who have been probably cured of their cancer, because they had early cancer and early treatment. Those patients should be told of their disease, because they can be enthusiastic disciples both for the physician and for the cause of cancer. They will carry the heartening and necessary word that cancer can be curable.

In the next few minutes I would like to discuss what can actually be done in the realm of palliative treatment for incurable cancer. Anorexia and malnutrition are probably the most common findings in patients who have incurable cancer. There are a number of ways to treat this anorexia and the secondary malnutrition. One can give a few units of insulin, alcoholic appetizers or beverages such as wine or cocktails, and so forth, about 20 minutes before mealtime, to stimulate the appetite. However, the beneficial effects are not consistent or great.

Dr. Delp and I have both given diuretics to patients with carcinomatosis of the abdomen. The diuretic will often materially decrease the amount of edema in the gastrointestinal tract, resulting in stimulation of the appetite. However, in my experience, the best method to stimulate appetite—to create an appetite so that the patient is voluntarily eating again—is forced feeding, usually by tube feeding.

The patient we saw earlier today demonstrates some interesting principles of stimulation of appetite. This lady has a large inoperable carcinoma of the pancreas. She was severely jaundiced, with pruritus. She had severe back pain. She had lost much weight and was grossly malnourished. She then had palliative surgical procedures which included alleviation of the biliary obstruction and the duodenal

obstruction. Despite this surgical palliation, however, she refused to eat for 30 days postoperatively.

There was no postoperative evidence of obstruction in any area. We felt that her anorexia and nausea were due entirely to poor liver function related to severe biliary cirrhosis. Her plasma proteins, which had been almost normal preoperatively, fell to 3.5 grams per cent, a low figure. She became edematous and appeared to be terminal in every sense of the word.

However, we instituted forced feeding and gave her by stomach-tube high supplements of protein digests. We also gave her whole blood intravenous transfusions as protein supplements. Intravenous injection of serum albumin proved to be too expensive. We also gave her fat emulsions orally—this can also be given intravenously if properly prepared. All of these things were given to furnish both proteins and calories to the patient.

The usual response sequence is that the patient will first begin to gain weight, then strength, and finally appetite. Dr. Elman has made an extensive study of this and has found that it may take a long time, depending upon the degree of malnutrition, for patients to regain their appetites. After about 30 days of tube feeding, this particular patient is now eating heartily, has gained strength, feels much better, and has a plasma protein level of 7 grams per cent, although she still has her cancer.

Pain is an important problem in incurable cancer patients. I want only to mention at this time that the use of nerve blocks, such as an intercostal nerve block or a somatic nerve block with procaine, may be extremely valuable. Many of my patients with incurable cancer have experienced relief of pain through the use of such nerve blocks, without having to receive morphine. Since many cancers have an area of infection or painful inflammation about them, antibiotics can be judiciously used to alleviate any pain related to these processes. Antibiotics should also be used to obviate the sepsis that often occurs in debilitated patients with incurable cancer. The many analgesic drugs available today can be rotated to retain their efficacy and to avoid the use of morphine and the consequent mental deterioration.

There are many other palliative measures available. Minor and even radical surgical procedures can be used purely for palliation. Hormones are extremely useful in palliation of some cases of incurable cancer of the breast or prostate. Irradiation, including rotation type of x-ray treatment, may be a useful palliative measure to reduce tumor size, obstruction, pain, or exudate formation. Radioactive iodine can reduce the size of primary or secondary tumors in some cases with cancer of the thyroid. Chemotherapeutic drugs, such as nitrogen mustard, may reduce tumor size or growth. Other simple measures such as paracentesis or thoracentesis, blood transfusions, the use of clean aesthetic dressings, and the administration of supportive drugs such as iron or vitamins are included in the literally thousands of things which the physician may use in treating the patient with incurable cancer.

The physician must justify the confidence and trust of the patient and his family that everything is being done which can reasonably be done to keep the patient comfortable and alive as long as possible. One must not build up false hopes within the patient about new developments or non-existent cures. However, the physician himself must always remember that conceivably during the period of palliative treatment, a dependable new and better palliative or even curative measure may become available. Our responsibility is always to do the best we can for these incurable human beings until they are overcome by death.

Dr. Helwig: Thank you, Dr. Friesen. Dr. Delp, would you continue this discussion from the internist's point of view?

Dr. Delp: Dr. Friesen and I have had numerous discussions, over a period of time, with regard to caring for the patient who has incurable cancer. I don't believe I have any real disagreement with any of his statements. However, I would like to reemphasize a number of things he has said.

First of all, there still is such a tremendous number of cancer patients who are incurable that cancer is now a public health problem. Because of that fact, I have objected to a good bit of the campaigning and publicity in the past five or ten years regarding the treatment of cancer. While I believe that some cancers are curable, particularly if caught early, I also believe that we have oversold the idea. Millions of people interpret this to mean that all cancer is curable. Practically every educated cancer patient hopes that his cancer is one of the curable ones and reasonably expects the medical profession to provide him with a cure. Such an optimistic cancer patient must make a great readjustment and may well suffer a severe psychological letdown when the physician somehow tries to explain to him that his cancer probably is not curable or that his chances of cure are rather remote.

On the other hand, optimistic publicity with the accent on early cures rather than on the general curability has helped in many instances to bring in the patient who would ordinarily be so frightened of the possibility of a diagnosis of cancer that he avoids his physician or the hospital. This apparently is done on the head-in-the-sand theory that the disease is not there until the definite diagnosis is made. It is the feeling of the American Cancer Society that their publicity and educational program

have helped to save a great number of lives which would have been lost if the patients had not been encouraged to seek early diagnosis and treatment. Admittedly, extra neuroses may be produced in patients who do not have cancer, as well as in some of those who do have cancer.

The second thing that I would like to reemphasize is that it is of prime importance to make the diagnosis as accurately and as exactly as it can possibly be made. As Dr. Friesen mentioned, this may entail getting and examining material from multiple biopsies. The physician must have real factual evidence before he can properly face the patient with the problem, give him some tangible picture of his disease, honestly discuss with him some intelligent plan of therapy or management, and offer him enough hope that he can face the future and even plan his affairs along some sort of schedule.

The third problem is the most thorny of all: how and when to approach and inform the patient of the diagnosis of incurable cancer. It is the one that is probably handled the most clumsily by the majority of physicians, and handled very crudely by a certain few physicians. I am firmly of the belief that all patients with cancer should be told that they have cancer. Those with curable cancer are easy to tell. Those with incurable cancer must be told with extreme care, and there are probably some situations in which it may be unwise to tell the patient at all.

Many people who are mentally ill, or already as ill from their emotional problems as they are from their cancer, are not apt to face the diagnosis of incurable cancer very well. However, none of us would face up to such a diagnosis very well. Nevertheless, there are some who simply cannot tolerate it, in which cases the physician might precipitate an explosive emotional crisis and suicide. If I actually thought that my patient might commit suicide immediately after being informed of such a diagnosis, then I think I'd be extremely hesitant in informing him at that time. But I feel that, as a rule of thumb, the patient should at one time or another be told exactly what he has.

Now you, as physicians, can do as some clinical scientists do: walk right up to the patient and slap him in the face with a death warrant by saying that he has incurable cancer. You can evade and sidestep the issue, for weeks at a time. You can lie to the patient in all sorts of ways. You can tell him in every manner except by blunt words that he does or that he doesn't have cancer. You can slide out of your responsibility by telling the relatives or the chaplain, leaving it to them to tell the patient, and blame them if anything goes wrong when the patient finds out. Or you can do it the right way: sit down alone with the patient, in the proper patient-physician manner, and confide in him, man to man, at an appropriate time.

If you have previously taken a personal interest in the patient, you will already have an idea of how to proceed. This should be done *alone* with the patient, with none of the relatives there. After all, you are the patient's physician, to whom he and the relatives have entrusted his welfare and his life. It may be done bit by bit, or you may discuss the problem with him at some length at one sitting. With any method, the news probably will be extremely painful to the patient. If you are a sincere and conscientious physician, it will likely be painful to you. However, after the first few minutes of such an interview have gone by in a kind and loving manner, you will usually find that the patient will begin to accept your information and prediction. After a week or ten days, the patient is usually much more easily controlled and much more able to face his remaining days on earth, knowing the diagnosis, informed of his chances, and still having some hope.

I don't think you should extend false hope of a cure to that patient who you know is rapidly facing the end, but you can give him hope for a somewhat prolonged existence. Three years ago I faced the problem of imminent death with a woman who was a schoolteacher in Tulsa. She had just had a biopsy of her liver and was found to have metastatic carcinoma in the liver. She had had a breast removed six years previously. The liver biopsy contained the only evidence that metastatic extension existed. Since this biopsy was in August and school began in a few weeks, it was necessary to face this problem expeditiously. We discussed it for some 45 minutes in just the manner outlined above and, much to my amazement, the patient got up, and said, "Well, I think I will go on down to Tulsa and teach. Do you see any reason why I shouldn't teach this next year?" I was a bit shocked, but managed to answer, "No, of course not." She not only taught that year in the schools in Tulsa, but she taught the following year too. The last few months of the second year she had considerable difficulty, requiring periodic abdominal paracentesis. Two months after school was out, she did finally succumb.

Her husband had been informed of the situation of course. Together they had faced the problem plainly, with no hiding and no subterfuges, in setting up their plans for their future existence together. That case illustrates what can be done under such trying circumstances if the patient is an adult, mature individual, able to face serious problems as he should.

In contrast to this case, I have a patient on the wards at the present time who has been informed roughly a dozen times within the past 18 months that she has an incurable cancer. This patient is immature and has no intention of ever accepting the diagnosis

of cancer. She has been to literally dozens of physicians, some of whom have unfortunately told her, "No, this is not a cancer, this is something else." As a matter of fact, she knows that she has a malignant lymphoma and repeats, "This is not a cancer I have, this is a lymphoma. This is not leukemia I have, this is a lymphoma." Since she absolutely refuses to accept the diagnosis of cancer, she has been unhappy and dissatisfied, with all of her complications and recurrences being similarly unacceptable to her. She represents a small group of cases in which even a skilled, patient, persistent psychiatrist may be unsuccessful in establishing contact with reality. Nevertheless, it's worthwhile to try, and most of the time the patient is benefited.

I believe that we are legally obligated to tell the patient he has cancer. I am quite sure that a physician could be haled into court, prosecuted, and even jailed or fined, if convicted, for not telling a patient he has cancer. I believe that it is a form of legal malfeasance and probably akin to malpractice not to inform them. Nevertheless, there are a few patients who cannot, for one reason or another, be personally informed. One always informs someone in the family, in any case.

Now, having squarely faced the problem of diagnosis and informing the patient, and having begun any definitive therapy possible, the physician should immediately outline the course of palliative therapy over a prolonged period of time.

I too am utterly opposed to giving patients narcotics except as a last resort. I would like to see their pain and discomfort controlled in any other way because I see dozens of patients every year who have been addicted to morphine months before they really had unbearable pain. They have become addicted to morphine usually because someone has said, "Well, the patient has cancer. My God, give him some morphine!" These patients become restless, unhappy, dissatisfied, frustrated, and just don't face their problem well. Few things will disturb a family more than a narcotic addict, and especially a narcotic addict who has an incurable cancer. There is no excuse for illogical or unnecessary use of narcotics in these situations. The giving of narcotics to cancer patients can be delayed, with a few exceptions, until a very late date.

I have found that various steroids can be useful in helping control pain. The more specific applications of testosterone and estrogen to patients with breast or prostatic cancer, of course, are well known, but I am referring to the non-specific uses. These steroids somehow have a non-specific pain-relieving quality and often give the patient a sense of euphoria. Cortisone is even more potent, although it cannot be used in patients who have large fluid collections in the body unless the fluid balance can be controlled by reasonable restriction of the salt intake. Some of my patients with rather serious and painful bone involvement have been given cortisone plus nothing else than one to three doses a day of Empirin compound, or A.S.A. compound containing a quarter grain of codeine, for as long as two years, to control their discomfort. The mechanism of these steroid actions is unknown to me.

Dr. Friesen has mentioned something else which I think is extremely important in the nutritional status of patients. This is the peculiar collection of edema fluid in the wall of the gastrointestinal tract with abdominal cancer, which may ruin the cancer patient's appetite and produce diarrhea and general indigestion. Most of the time this problem can be managed rather simply, by using any one of the host of effective diuretic agents now available. Diamox is one of the more harmless diuretics to use in this situation.

The medical profession must see to it that for each individual case there is a physician who will take the ultimate personal responsibility for the patient who has an incurable cancer. There is too much of a tendency to shunt this responsibility from one to the other. It's too easy, for example, for physicians in the so-called ivory tower of a large medical center to see a patient with an incurable cancer for a matter of a week or ten days and then shunt him off to somebody else.

It has sometimes been argued whether the surgeon or the internist is best qualified to take care of such patients. Obviously the one best qualified to take care of the patient who has incurable cancer is the individual physician who personally knows the individual patient best, has the best rapport with the patient, and can actually keep the patient's confidence. Taking care of a patient with incurable cancer is one of the situations which clearly separates those physicians who are personally dedicated to the complete welfare of their patients from those who are superficially motivated. We must keep our obligation to our patients with cancer. There must be a good reason for sending the patient to a nursing home or transferring him to someone else's hospital service—this must not be done just to get the patient out of your way. If your patient goes to his home, or to a nursing home, he's still your patient and your responsibility.

There have been cases where I have realized that I was less qualified than someone else for taking care of a patient with cancer. For instance, on one occasion I requested a surgeon, who I knew had much better rapport with the patient than I did, to take over the care of that patient, or at least to help me. So we took care of the patient together, although there was no longer anything about that patient that represented a surgical problem. It was simply a situation in which

the physician with the best contact received the greatest degree of confidence from the patient and was therefore best able to ameliorate her symptoms.

I feel rather intensely about a good many of these problems, but I believe the physician has to have convictions for desperate situations. Whatever your feelings, there is no doubt that your management of these unfortunate patients will be much easier and much more effective if you sincerely do everything humanly possible for the patient and treat him in every way as a fellow human being.

Dr. Helwig: Thank you, Dr. Delp. I would like to emphasize how difficult it may be sometimes to predict correctly the patient's reaction to the diagnosis. For example, during the ten years when I was actively engaged in taking care of cancer patients, I had two sobering experiences which pointed up the grave difficulty in evaluating suicidal tendencies. Within a period of one month, and at the tearful request of the wife in each instance, I was asked to tell two patients that they had incurable cancer. I had seen both patients for months and felt I knew them both well.

The first fellow, with carcinoma of the jaw, was carefully told in the course of events that he had about a 20 per cent chance for a five-year arrest. He broke into tears, then laughed, and then thanked me. "My God, Doctor, why didn't you tell me this a year ago? I would have been happy if you'd told me. I'll never forgive you. But now you've told me, I'm resting at ease." Thirty minutes later his wife called me with the information that he had gone into the bathroom and slit his throat with his straight-edged razor.

The other chap was a well known druggist in this local area. He had been treated for carcinoma of the tongue with cervical metastases by a radiologist in Philadelphia. A recurrence developed in the overly irradiated zone in his neck. We got a biopsy, and I was going to soften the outcome by telling him, "It's just a little granulation tissue." However, he and his wife independently convinced me that he really wanted to know the truth and was stable and mature enough not to react violently. After we had carefully discussed the problem, he said, "Thanks, Doctor, I knew it was. Thanks very much." A few minutes later he pulled a gun from his desk drawer and put it to his head.

Since then, I have always been hesitant to be too frank with these people. I believe that one can help them adjust gradually to the realization of their disease without telling them the whole story at any one time. Certainly no one wants to be an executioner. We must remember too that certain people who might have lived happily in blissful ignorance until a later time for facing the problem, might become increasingly despondent or psychotic and gradually sink deeply into a vegetative depressed existence after being told, even though an explosive reaction had been avoided. Certainly the family should always be informed of the diagnosis, even though this means that they must play a masquerade if the patient is not to be told until later.

We should reemphasize Dr. Delp's point that, in certain cases, the physician may be liable to being sued for not informing the patient. One physician in Colorado who had operated on a wealthy businessman had told only the family that the man had cancer of the prostate but might live for five years or even longer. The businessman sued and collected from the doctor because he said that he would have made different business arrangements for his future, had he known the story.

Some of the physicians at a large cancer hospital in New York City have adopted the approach of evading direct answers to the patient's questions and apparently find that eventually the patients all make their own diagnosis and adapt in one way or another.

Dr. Delp: I can certainly sympathize with the two unhappy situations which Dr. Helwig has mentioned. I've been facing the problems of suicide and others for 20 years now and have had eight patients who did fool me about their stability and commit suicide. They were, however, all new patients, and none of them had cancer. I have been lucky enough to face the problem of incurable cancer with several hundred patients in the personalized manner I have discussed above, and I have not yet had a single cancer patient who committed suicide.

I will still continue to tell all my patients with incurable cancer what they have and what it means. This will be done as painlessly as possible. Each physician will have to make up his own mind. I would certainly disagree with the unforgivable attitude of the group in New York who just sort of pass the problem off. I would wonder how many of their uninformed patients literally hungered for a chance to confide in some personally dedicated physician to ease their minds on many aspects of their problems.

Dr. Williamson: The neurosurgeon should be one of the last resorts in the palliation of pain in patients with incurable cancer. I certainly agree that the surgeon, internist, and radiologist should do all that they can first. We are concerned with those few patients with cancer who still have unbearable, intractable pain, in spite of the internist's ability, devotion, and drugs, the surgeon's palliative operations, or the radiologist's symptomatic irradiation of the tumor mass. Severe pain may be produced, for example, by a malignant process invading the nerve roots of the lumbosacral plexus, or around visceral nerves as in the pancreas. The physician's duty to the patient in such a case is still that of rendering com-

fort by some means or other. I heartily agree with Dr. Friesen and Dr. Delp that narcotics are not the answer.

When we, as neurosurgeons, see patients who have had the skillful care of a good doctor, including all sorts of palliative measures, and are still suffering with unbearable pain, we study them carefully to see whether we can relieve their pain and stop their suffering by interrupting certain nerve pathways by one method or another. We will not attempt to go into detail on any of these methods but will emphasize rather the various neurological areas where one can interrupt nerve pathways to give relief of pain.

First, the use of local anesthetic nerve blocks, as already mentioned by Dr. Friesen, constitutes temporary symptomatic treatment only. By the time the neurosurgeon sees these patients, they need desperately some permanent measure to relieve their intractable pain. Rarely do we permanently interrupt the continuity of a peripheral nerve for relief of pain. For one reason, the cancer is usually not peripheral, such as out in an extremity fed by only one major nerve. Secondly, if the patient's pain is in an extremity such as a hand or a foot, severing the brachial plexus or the sciatic nerve would render that extremity utterly useless. An anesthetic hand is a useless one, and an anesthetic foot is soon going to have decubitus ulcers from walking on it. One might better amputate the whole limb. This is not a practical approach to the relief of pain.

In the region of the cauda equina, we sometimes will do another last-resort procedure which I mention only to clarify one point. In a patient with intractable pain due to invasion of the lumbosacral plexus by carcinoma of the pelvis, we will sometimes inject intrathecal alcohol, just to obliterate the cauda equina functionally. Let me emphasize that this procedure is reserved only for that permanently bedridden patient who will never walk or void again because he is terminal, or because his lumbosacral plexus has been partially obliterated by cancer or by surgery, and who is suffering from unbearable pain in the pelvis or lower extremities. The procedure should be done with the patient's buttocks elevated. The alcohol will rise when injected into the spinal canal, since it is lighter than the spinal fluid, and will float around the cauda equina to anesthetize the legs and knock out the pain. Do not inject alcohol into the spinal canal of any patient that you expect to walk again. You may lay yourself open to prosecution and suit.

A procedure for relief of pain at a higher level is the so-called rhizotomy. When the nerve roots enter the spinal canal, the sensory components enter the dorsal or posterior roots. We can do a laminectomy and cut only sensory roots, so that we do not produce any motor paralysis, a so-called sensory rhizotomy. This procedure is not often practical, because cancer growing in a region such as the inguinal region to produce inguinal pain might involve nerve endings which connect through roots all the way from T-10 down to S-5. Such a situation would require widespread laminectomy and the probable creation of numbness of a large area of a leg, genitals, and abdomen. This would necessitate much too radical an operation. Rarely is the pain from cancer so focal that a discrete rhizotomy is the answer. Moreover, a rhizotomy which produces an anesthetic extremity to relieve pain is not advisable.

A better procedure is the cutting of nerve tracts in the spinal cord where pain and temperature sensations cross over and ascend in the spinothalamic tract of the opposite side. The neurosurgeon can insert a knife into the spinal cord at the proper level, swing around laterally, and obliterate the lateral spinothalamic tract, to produce loss of pain and temperature sensations on the opposite side from this level down. A successful chordotomy at T-1 should produce loss of pain and temperature perception on the opposite side of the body, from about T-5 down. It thus would *not* destroy the sense of touch or proprioception, or produce decubiti, or weakness and dysfunction of the bladder. If technically successful, it produces a permanent loss of pain. This is probably the best neurosurgical approach to pain, although the results are sometimes debatable.

There are still higher levels at which one can operate. A spinothalamic tractotomy can sometimes be done in the brain stem to relieve pain due to cancer of the neck and shoulder regions. That isn't very practical and is still somewhat experimental. As you know, the pain fibers come on up through the brain stem to the thalamus and then back to the postcentral gyrus. Resection of the postcentral gyrus is done occasionally for phantom limb, with mediocre results because it does not always abolish pain.

The prefrontal lobotomy offers one other interesting surgical approach to pain. There is an element of pain that has to do with the patient's anxieties, his fears, and his reactions to the pain. There are two parts to pain: the pain sensations which the patient actually feels, and the way he reacts to the pain. The prefrontal lobotomy takes away fear and anxiety about pain and thus changes the way the patient reacts to his pain. Patients who have a lobotomy still have their actual pain but will comfortably sit there reading a newspaper, talking small talk, or smiling, and not bring up the matter of pain or ask for hypos. If you ask, "Do you still hurt?" they reply, "Oh, yes, but look here what's in the paper today." That is their attitude.

It makes me personally feel rather queer to see those people and to think of what we have done so that they will no longer complain of pain. It is hard

to tell whether the operation has somehow raised the threshold against pain or whether the fears and anxiety reactions hurt more than actual pain. Our series of lobotomies has been relatively small. Some neurosurgeons do a lot of unilateral lobotomies with relief of pain and narcotic addiction for four or five months, until the pain comes back again. Bilateral lobotomies create rather serious personality defects, which in some cases, however, may be preferred by the patients to their pain.

Now let's reevaluate all these procedures. I would first like to say that as a neurosurgeon my most difficult problem is to evaluate the patient's pain. I don't think that the neurosurgical specialist who briefly walks in to see the patient and recommends a chordotomy can adequately evaluate that patient for chordotomy. As Dr. Delp has said, one has to know the patients and their reactions to pain. I don't know that I can ever tell how much patients are really hurting, for I cannot tell how much of their pain is real and how much is their reaction to pain.

Most of the patients we've seen are sent in to us late, by which time they are often long-term narcotic addicts, and the operation can ofttimes be expected to be unsuccessful on that basis alone. One can do a technically nice chordotomy, but, because of the narcotic addiction, the patient often continues to complain of the operative wound, or of radiation of pain at the level of the chordotomy, simply to continue to get his narcotics. Whether or not the operation is successful depends a great deal on the personality of the patient. By the time so many of them come to us, pain is their whole life. They are wrapped up in this pain—it furnishes them with a necessity for much care and attention and narcotics.

So neurosurgery has no panacea to offer all of these patients with painful incurable cancer. However, there is a select group of patients who, in spite of all other measures, still have pain; certainly, in preference to making them addicts, a neurosurgical procedure might be offered.

There are apparently some technical reasons for failure of chordotomy. There are patients on whom nice operations have been done, but several weeks later their sensory level recedes and their pain comes back. In some cases rhizotomies fail, in that areas of total anesthesia are produced but pain still hurts the patient. And who are we to know how much the pain is really hurting someone else?

Some famous neurosurgeons are advocating doing these operations prophylactically, before cancer patients get their pain. I can't agree with that, for the operations are not so simple or so successful as to justify such optimism. Neither do I believe that we should do a chordotomy, or any other neurosurgical procedure, in a patient who is terminal or dying. The patient ought to have at least a reasonable life expectancy, say of roughly six months, as well as can be estimated. If a patient is bedridden and expected to die within a couple of months, then I believe that medical measures of any sort, even narcotics, are better for the patient than having a major operation which can never get him back on his feet and which will be the source of some discomfort until death.

In summary, I find myself unable to predict whether or not an individual patient is going to be substantially relieved of pain by these procedures. Certainly if the patient has obvious reason to be suffering unbearably and if everything else has failed, then I do think we have something to offer which many times has produced grateful patients.

Dr. Helwig: Thank you, Dr. Williamson. I wonder if you can give us some of the radiologist's ideas on today's discussion problem, Dr. Germann.

Dr. Germann: I'd like to say that I've learned a good bit sitting here today and have been enjoying this session. I have certainly used radiation therapy often just for palliative control of pain, and sometimes it does very well. Irradiation over bone metastases, as well as involved viscera, may relieve pain considerably. Sometimes, however, the radiation sickness which occurs after broader irradiation makes you wonder if it's worth it. You have to observe and decide as you go along; if the patient is getting radiation sickness, stop irradiation and go to other methods. Recently we have been using radiogold and radioiodine, particularly gold, for effusions due to cancer. About 50 per cent of the patients get pretty good relief; others are not helped a great deal, if at all. These simply constitute additional tools for palliation of incurable cancer.

Dr. Helwig: I think perhaps it would be well justified to emphasize again that the diagnosis of malignancy must first be absolutely established, as well as its stage of incurability. Not too long ago a doctor's mother-in-law was having severe gastrointestinal hemorrhages. The x-rays appeared to reveal a typical advanced cancer of the stomach. With such a presumptive diagnosis, she was advised to have no surgical procedure done. At the autopsy it was found she had a large resectable benign myoma of the stomach.

In another case, a woman in the cancer age group of 50 years or so had ascites, with bilateral hard pelvic masses which were presumptively diagnosed as carcinoma of the ovary. However, someone decided that the case should not be considered hopeless until explored, and they found that she had only a Meigs' syndrome.

In still another case, a woman did have bilateral carcinoma of the ovary with ascites and innumerable implants on the pelvic peritoneum. Her pelvic viscera were removed and she is still alive now, 18 years

postoperatively! So never make a diagnosis of hopeless malignancy unless you have the absolute biopsy evidence.

Furthermore, don't make wild guesses as to survival—many people will live a long time with ultimately hopeless cancer. I've seen men with cancer of the prostate live 10 years, and I've seen other patients live with their inoperable cancers 5 and 6 years. I remember well one woman whom we opened up, only to find the abdomen full of grapefruit-sized cystic masses from carcinoma of the ovary. She was sewed up, went back home, and did her own housework for 12 years before succumbing. Every surgeon and internist with a wide experience with cancer has had cases where the tumor had been actually removed or destroyed but were called incurable because the physicians felt that they had not been able to get out all the cancer.

So when we tell the patient that he has an incurable cancer, we want to be mighty sure of ourselves. There may be a right time and a right manner in which to tell the patient, but you'd also better have the right information! The man whom you misinform that he is doomed to die in 18 months may not only live to haunt you 10 years later, but he is not apt to have enjoyed his extra unexpected years of postponement.

BIBLIOGRAPHY

1. Kelly, W. D., and Friesen, S. R.: Do cancer patients want to be told? Surgery 27:822-826, 1950. (See also editorial: Should patients be told they have cancer? Surgery 27:944-947, 1950.)
2. Shall the patient be told? Monthly Bull. Indiana Bd. of Health 54:58-59, 1951.
3. Ginsberg, R.: Shall the elderly cancer patient be told? Geriatrics 4:101-107, 1949.
4. Bowen, O. R.: Should patients be told when the diagnosis is cancer? J. Indiana State M. A. 46:303-309, 1953.
5. Fitts, W. T.: What Philadelphia physicians tell patients with cancer, J.A.M.A. 153:901-904, 1953.
6. I live with an inoperable cancer, Today's Health 29:22-23; 60, 1951.
7. Bellak, L.: Psychology of physical illness, New York, Grune and Stratton, Inc., 1952.
8. Emotional aspects of cancer (seven references). The Cancer Bulletin 5:4-6, 1953.
9. Some problems of a family physician in cancer, Nassau Med. News 24:6-7, 1951.
10. Leibowitz, S.: The conduct of the internist toward the patient and his family when a diagnosis of malignancy is established, N. Y. J. Med. 51:1421-1424, 1951.
11. Cameron, C. S.: Cancerophobia, J.A.M.A. 147:526, 1951.
12. Cooper, G.: The management of incurable cancer, South. Surgeon 15:132-137, 1949.
13. La Due, J. S.: The management of terminal patients with inoperable carcinoma, J. Kansas Med. Soc. 54:1-6, 1953.
14. Finesinger, J. E.; Shands, H. C., and Abrams, R. D.: Managing the emotional problems of the cancer patient (19 references). CA-Bull. Canc. Prog. 3:19-31, 1953.
15. Morgan, H. J.: The care of the patient with terminal cancer, Rocky Mtn. Med. J. 45:116-119, 1948.
16. Eliasberg, W. G.: Psychotherapy in cancer patients, J.A.M.A. 147:525-526, 1951.
17. Keller, C.: Raising the cancer patient's morale, Am. J. Nursing 49:508-511, 1949.
18. Lofthouse, E. M., and Stokes, G. A.: Nursing care of children with cancer, Am. J. Nursing 53:415-418, 1953.
19. Sutherland, A. M., and Orbach, C. E.: Psychological impact of cancer and cancer surgery, Cancer 6:958, 1953.
20. Shands, H. C.; Finesinger, J. E.; Cobb, S., and Abrams, R. D.: Psychological mechanisms in patients with cancer, Cancer 4:1159-1170, 1951.
21. Butler, B.: The use of hypnosis in the care of the cancer patient, Cancer 7:1-14, 1954.
22. Murphey, B. J.: Psychological management of the patient with incurable cancer, Geriatrics 8:130-134, 1953.
23. Orbach, C. E.: Psychological aspects of cancer patient care, Phila. Med. 49:526-528, 558-559, 1953.
24. The cancer patient's relatives, Cancer Bull. 5:92-93, 1953.
25. Ford, J. C., and Drew, J. E.: Advising radical surgery: a problem in medical morality, J.A.M.A. 151:711-716, 1953.
26. Livingston, W. K.: What is pain? Sci. Am. 188:59-66, 1953.
27. Turnbull, F.: Treatment of pain in malignant disease, Vancouver M. A. Bull. 29:302-305, 1953.
28. Grantham, E. G., and Spurling, R. G.: Selective lobotomy in the treatment of intractable pain, Ann. Surg. 137:602-608, 1953.

Urological Meetings in Kansas City

A series of meetings on urological subjects has been announced for the 1954-1955 season by Dr. Clarence S. Capell, secretary of the Kansas City Urological Society. Each meeting, to be held at the Pine Room, Fred Harvey Restaurant, Kansas City Union Station, will consist of a social hour at 6:00, followed by dinner, a scientific paper, and the presentation of urograms. The following programs will be given:

September 15—The Clinical Significance of Renal Efficiency—W. M. Valk, M.D., and R. H. Owens, M.D., Kansas City.

October 7—Uretero-Enterostomy—J. J. Cordonnier, M.D., St. Louis, and A. L. Stockwell, M.D., Kansas City.

November 17—Tumor of the Bladder—A. G. Isaac, M.D., Newton, and Pratt Irby, M.D., Fort Scott.

January 19, 1955—The Female Urethra—R. L. Hoffman, M.D., and William M. Leifer, M.D., Kansas City.

February 16—Untoward Reactions from Urogram Injections—J. G. Siceluff, M.D., Springfield, and B. W. Andrews, M.D., Kansas City.

March 16—Urinary Calculi—H. E. Carlson, M.D., and J. Zellermayer, M.D., Kansas City.

April 24—Urogenital Anomalies—O. W. Davidson, M.D., and R. Bristow, M.D., St. Joseph.

The last hour of each session will be devoted to the presentation and interpretation of urograms. All urologists in the area are invited to attend and to take films.

Birth Rate Rises

The national birth total in the first four months of 1954 topped the same period of 1953 by about 30,000, according to vital statistics estimates released last month by the Public Health Service. Births all through 1953, estimated at 3,971,000, broke all previous records. This gave a rate of 25.1 births per thousand population.

Marriages, however, have continued to fall this year, after sinking in 1953 to 9.7 marriages per thousand population, the lowest annual rate since 1933. This is attributed to the fact that relatively few young people have been reaching marriageable age because of the low birth rates of the 1930's.

ACTIVITIES OF MEMBERS

Dr. Edward X. Crowley was elected president of the newly organized Wichita Obstetrics and Gynecological Society. Serving with him are Dr. E. G. Anderson, president-elect; Dr. J. S. Menaker, vice-president, and Dr. Grant E. Evans, secretary-treasurer.

June 27 was designated as "Dr. Floyd Eldridge Wallace Day" by the city council of Chase, honoring the physician on his 50th anniversary in the practice of medicine. During the day's program, sponsored by the Lions Club and the V.F.W., a gold watch from the Rice County Medical Society was presented by Dr. R. E. Bula.

Dr. Z. H. Snyder, 84, who began practice in Palmer in 1903 and has practiced in Greenleaf since 1905, was guest of honor at a potluck supper at the Greenleaf Community Clubhouse, June 13. Now retired, Dr. Snyder will make his home in Hays.

Two Wichita physicians, Dr. Eugene G. Anderson and Dr. Lawrence E. Woodard, recently became diplomates of the American Board of Obstetrics and Gynecology.

Dr. Floyd L. Smith, Colby, addressed the Colby Rotary Club recently on the subject of a possible connection between cigarette smoking and lung cancer.

Dr. Mary Glassen, Phillipsburg, who is now touring Europe, appeared on the "Welcome Travelers" television program before she sailed from New York. Gifts presented to her in connection with the TV appearance were for the home for exceptional children in Topeka. In Europe she plans to attend a meeting on gerontology and a program on obstetrics and gynecology, in addition to sightseeing in England, Holland, Belgium, Italy, and France.

Dr. John R. Kline, Wichita, spoke on "Radioactive Isotopes in Medicine" at a recent meeting of the Sedgwick County Medical Assistants' Society.

Dr. Victor H. Hildyard, who has been practicing in Baldwin since 1945, moved to Minneapolis on July 1 to begin a residency in surgery at the University of Minnesota Medical School. Dr. Raymond L. Pendleton, formerly of Lawrence, has taken over Dr. Hildyard's practice in Baldwin.

Dr. J. W. Parker, Burlington, is now serving as president of the Coffey County Medical Society. Dr. A. B. McConnell, Burlington, is secretary.

Dr. George W. Jackson, director of institutions for the Kansas State Board of Social Welfare, was recently certified by the American Psychiatric Association as a mental hospital administrator. This certification is based on training and experience over and above that required for certification by the American Board of Psychiatry and Neurology.

Dr. Ray T. Parmley, Wichita, was recently elected secretary-treasurer of the Southern Society of Anesthesiologists.

Dr. I. Clark Case, who has been clinical director at Topeka State Hospital since 1951, resigned on July 1 to join the department of adult psychiatry at the Menninger Foundation, Topeka.

Dr. Rene M. Gouldner, Wichita, recently returned from a trip around the world by plane. Feature stories about his travels were printed in the *Wichita Eagle* and the *Wichita Beacon*.

Dr. E. W. Christmann, who has been practicing in Manhattan during the past 16 months, moved to Wamego recently and opened an office there July 1.

Dr. Chester M. Nelson, Oberlin, announces that Dr. James Kauffman, formerly of El Dorado, is now associated with him in practice. Dr. Kauffman, a graduate of the University of Kansas School of Medicine, is a veteran of the Korean War.

Dr. Howard E. Snyder, Winfield, has been elected to membership in the Committee on Trauma, American College of Surgeons.

Dr. G. F. Helwig, Topeka, closed his office recently to move to San Jose, California, for a two-year residency in anesthesiology.

Dr. George J. Millet, who has been taking postgraduate work in surgery in Buffalo, New York, during the past year, has resumed his practice in Larned in association with Dr. LeRoy Shepard and Dr. S. T. Coughlin.

Dr. Maurice S. Wessell, who has practiced in Hiawatha during the past three years, closed his office there on July 1 and has opened an office in Burlington.

Dr. Hugh R. St. John, Concordia, observed his 40th anniversary in the practice of medicine last month.

Dr. Charles E. Stevenson, Neodesha, has completed a month's postgraduate study of surgery at the Cook County Graduate School of Medicine, Chicago.

Dr. Robert W. Blackburn, who has been studying surgery at the VA Hospital, Albuquerque, New Mexico, during the past year, has returned to his practice in Council Grove.

The Gelvin-Haughey Clinic, Concordia, announces that Dr. Marion Pearson is now a member of its staff. Dr. Pearson, a graduate of the University of Kansas School of Medicine, is a veteran of the Korean War and recently completed a surgical residency at General Hospital, Kansas City, Missouri.

Dr. Harry Lutz, Augusta, has been appointed director of the health department for Butler and Greenwood counties.

Dr. A. C. Armitage, Hutchinson, announces that Dr. J. L. Perkins is now associated with him in practice. Dr. Perkins is a graduate of the University of Kansas School of Medicine.

Dr. George Hopson announced the closing of his office in Plains on July 15 and is moving to Richland, Missouri, to practice there.

The Ashley Clinic, Chanute, announces that Dr. Donald E. Ray, a 1953 graduate of the University of Kansas School of Medicine, is now on its staff. Dr. Ray completed his internship at St. Joseph's Hospital Kansas City, Missouri, last month.

Dr. Alexander C. Mitchell, staff physician at Watkins Hospital at the University of Kansas, Lawrence, for two years, resigned that position recently to be in private practice in Lawrence in association with Dr. H. Penfield Jones and Dr. G. E. Manahan.

Dr. M. J. Cox, Dodge City, addressed the Cimarron Rotary Club last month on the subject of cancer. He also showed the film, "The Warning Shadow."

Dr. Chauncey G. Bly, of the Department of Pathology and Oncology, University of Kansas Medical Center, went to South America last month to attend the Sixth International Cancer Congress in Brazil.

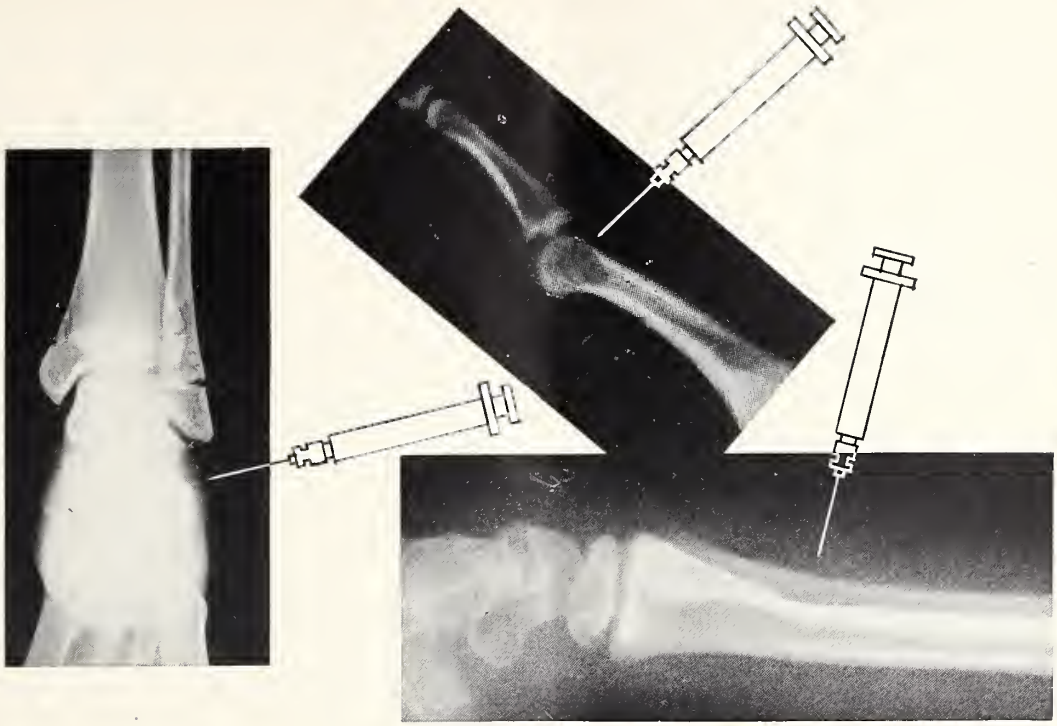
Dr. Edward D. Greenwood, Menninger Foundation, Topeka, is attending an international conference on mental health in Toronto, Canada, this month. He is a participant in one of the panel discussions.

Dr. William H. Browning, Wichita, was speaker at a Tri-County Medical Society meeting held at Wellington on June 24. His subject was "The Conservative Surgical Management of Hydronephrosis."

Three physicians became members of the Marion County Society at a meeting held last month. They are Dr. Robert D. Wood and Dr. Royal A. Barker, Peabody, and Dr. Roland L. Krause, Goessel.

Dr. H. B. Vallette, Beloit, announces that Dr. Ralph R. Reed is now associated with him in practice. Dr. Reed, a 1953 graduate of the University of Kansas School of Medicine, completed his internship on July 1 at the U. S. Naval Hospital, Oakland, California.

Dr. James Cuthbertson, who practiced in Sterling before going to San Mateo, California, for a residency, has announced plans to begin practice in Cupertino, California.



Use of Alidase® in Closed Wounds: Contusions, Sprains, Dislocations, Simple Fractures

In traumatic surgery[1] where "definitive treatment . . . is often delayed while the surgeon waits for nature to dispose of hematoma and oedema" Alidase is an efficient means[1, 2] of accelerating dispersion of accumulated fluids.

Swenson[2] has described his highly successful results with Alidase in various types of closed wounds. He summarized them as follows:

To remove local fluid accumulations in contusions or bruises, "The usual dose, 500 viscosity units Alidase® mixed in a small amount of normal saline, is injected into the localized fluid. Mixing the hyaluronidase in 1 per cent procaine solution will also produce local vasodilatation, relief of local pain and more rapid absorption of the fluid mass. This method can also be applied to traumatized bursae or synovial spaces which do not respond to repeated aspirations."

The point of maximal pain is infiltrated with 10 cc. of a 1 per cent procaine solution to which 500 viscosity units of Alidase have been added. With this simple technic, a high percentage of successful results has been obtained.

Alidase may be used to advantage to produce more rapidly a short-acting, complete block anesthesia and to facilitate reduction in subluxation or complete dislocations of the interphalangeal joints. When anesthesia is required for fracture reduction, local block anesthesia can be simplified by adding Alidase to the anesthetic solution. Alidase also tends to decrease local edema and hematoma formation.

Fluids administered with Alidase are rapidly absorbed from subcutaneous tissue. The simplicity of hypodermoclysis avoids the cumbersome arm board, permits convenient administration with little or no pain or swelling, is vein-sparing and saves nursing time in such conditions as burns, postoperative states, toxemias and parenteral alimentation.

Alidase (brand of hyaluronidase) is supplied in serum-type ampuls of 500 viscosity units. It is accepted by the Council on Pharmacy and Chemistry of the American Medical Association. G. D. Searle & Co., Research in the Service of Medicine.

---

1. MacAusland, W. R., Jr.; Gartland, J. J., and Hallock, H.: The Use of Hyaluronidase in Orthopaedic Surgery, J. Bone & Joint Surg. *35-A*:604 (July) 1953.
2. Swenson, S. A., Jr.: Minor Surgical Aspects of Closed Wounds, Am. J. Surg. *87*:384 (March) 1954.

St. John's Hospital, Salina, announces that Dr. Donald Wald is now radiologist on its staff. Dr. Wald was graduated from the University of Kansas School of Medicine in 1945 and spent four years in graduate work at Philadelphia General Hospital. He has been practicing in San Antonio during the past two years.

Dr. Roland D. Brooks, who was graduated from the University of Kansas School of Medicine last year and completed his internship at Madigan Army Hospital, Tacoma, Washington, on July 1, has announced the opening of an office in Madison.

Dr. W. H. Algie, Kansas City, was named president of his local city-county board of health last month. Dr. LeGrand Byington was reelected secretary.

Dr. Robert R. Snook, who has been practicing in McLouth for six years, was the subject of a feature story in the *Topeka State Journal* on July 10.

Dr. William Tappen, who interned at Bethany Hospital, Kansas City, after completing his studies at the University of Kansas School of Medicine, is now practicing in Atwood in association with Dr. E. T. Gertson.

BLUE SHIELD

Additional Blue Shield Benefits

The Blue Shield Board of Trustees has approved the following additional benefits with no increase in membership dues. These changes will be effective October 1, 1954.

A. Benefits Increased

1. Surgical Procedures:

Fracture of Tibia from $55 to $60

Submucous Resection from $55 to $65

2. Medical Service (non-surgical cases in the hospital): Beginning with the 31st day through the 90th day—increased from $2.00 to $3.00 per day (members age 65 and over or treatment of mental and nervous disorders will not be entitled to service beyond the 30th day in accordance with provisions of the regular Blue Shield Agreement.)

3. Anesthesia Payments: Anesthesia payments in connection with the following surgical procedures have been increased as per schedule below:

	Old Fee	*New Fee*
Cholecystectomy	$12.50	$15.00
Common Duct Exploration	12.50	17.50
Radical Mastectomy	12.50	15.00
Coarctation of Aorta	——	25.00
Nephrectomy	12.50	15.00
Prostatectomy	12.50	17.50
Thyroid Lobectomy	12.50	15.00
Bilateral Subtotal Thyroidectomy	$12.50	$17.50
Cesarean Section	12.50	15.00

B. New Benefits

1. Medical Services:

(a) *Massive Exchange Transfusion of Blood.* Payment will be made for an exchange transfusion of at least 350 cc. for treatment of erythroblastosis fetalis, fee $60.

(b) *Additional Care for Newborn.* In the case of premature infants (weighing less than 4½ pounds at birth) or critically ill newborn (regardless of weight), payment will be made beginning on the basis of the usual non-surgical care in the hospital. No payment will be made for the first or last day.

(c) *Intensive Medical Care.* For cases requiring medical care (such as diabetic coma, comatose state, severe coronary, etc.), up to $50 will be paid in addition to the regular payment. These cases will be submitted on a separate form when the doctor thinks the case qualifies, and they will be handled by the Blue Shield Review Committee on an individual basis. Special forms for submitting intensive medical cases will be furnished by Blue Shield.

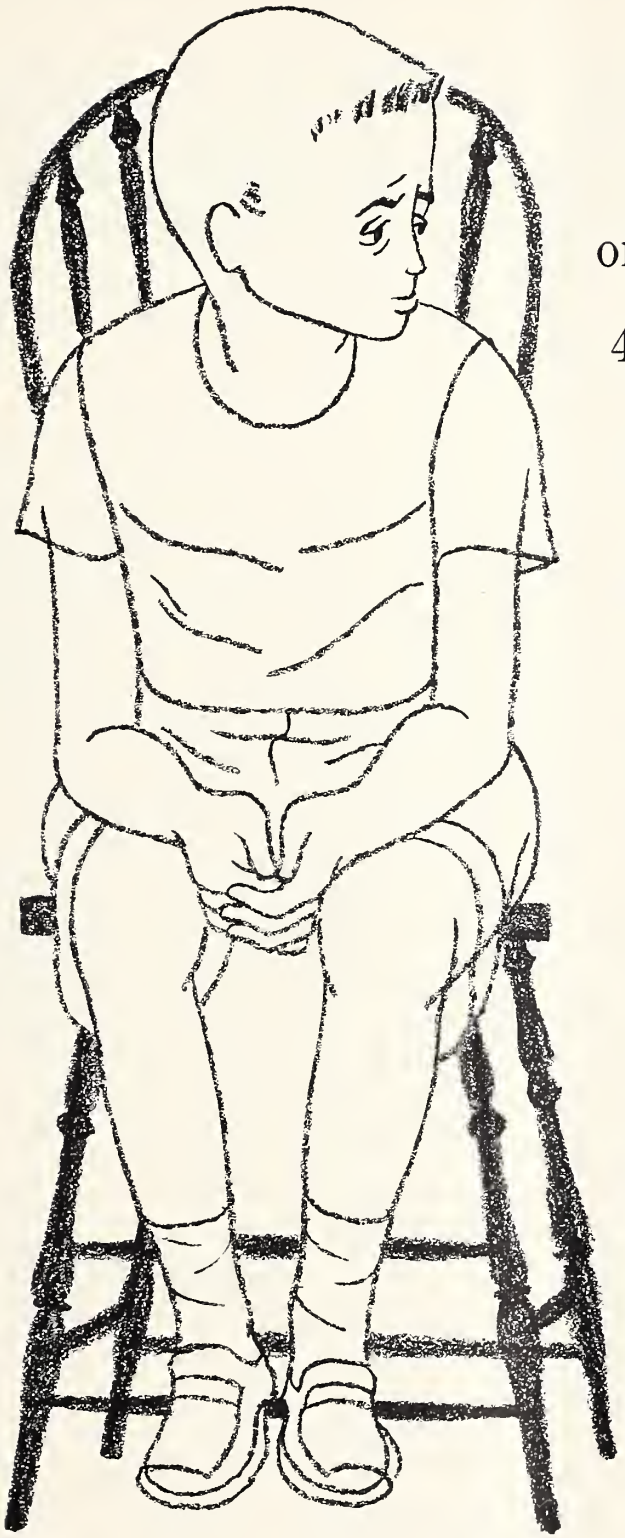
The intensive medical care benefit is an experimental venture and will *not* be a part of the contract. If it works out satisfactorily, it will be added later.

Additional benefit riders, along with proper explanation and necessary supplies, will be sent to all participating physicians by September 1. These additions will also be in the new *Physicians' Manual* which will be ready for distribution about October 1.

These increased benefits are the result of recommendations made by doctors participating in Blue Shield through their Fee Committee. Although all the recommendations could not be accepted at this time because of cost, this is one more effort toward a better Blue Shield contract.

At one time almost all children in New York City had been infected by the tubercle bacillus before they finished grammar school; now only about 10 per cent are found to be reactors to the tuberculin test by 12 years of age.—*Haven Emerson, M.D., NTA Bulletin, May 1954.*

pediatric preoperative sedation



one of the
44 uses for
short-acting

NEMBUTAL®

"**A** barbiturate which seems to have a most consistent effect in my experience is NEMBUTAL (*Pentobarbital, Abbott*) . . . administered one hour before operation and morphine sulphate twenty minutes before the patient goes into the operating room.

"If this preoperative medication is followed, the child will not be apprehensive and will often require less than the usual amount of anesthetic . . . one is impressed with the quiet sleep they produce and more impressed with the quiet uneventful recovery and infrequent nausea and vomiting." Abbott

Schaerrer, W. C., J. Missouri M. A., 37:287.

408152A

Physiological Theories of the Etiology of Alcoholism

Robert D. Parman, M.D.

Kansas City, Missouri

"What wonders doth wine! It discloses secrets, ratifies and confirms hopes, thrusts the coward forward to battle, eases the anxious mind of its burden, instructs in the arts. Whom has not a cheerful glass made eloquent, whom not quite free and easy from pinching poverty."—Horace

Why are we faced today with problems concerning a magic potion which according to Horace has such wonderful properties; why is it we should inform ourselves as thoroughly concerning the problem of alcoholism as we do concerning tuberculosis? It is because there are actually more alcoholics than patients who have recognized active tuberculosis. Because of the nature of the disease, its difficulty in management, its evasive etiology, and its possible moral implications, we often veer around this problem.

An alcoholic may be defined as a person who is addicted to alcohol; addiction is defined as that condition of the mind or body induced by some drug without which the mind and body suffer serious derangements.[1] In literature on alcohol addiction it is not always clear whether this strict definition and understanding of alcoholic addiction were used, but it is assumed so.

The incidence of alcoholism varies from author to author, some placing its incidence at 750,000;[30] others, like Smillie[2] in his public health text, say that 6 to 7 per cent of our total population find alcoholism a problem at some time during their lives. Jellinek and Kellen[3] estimated the total number of alcoholics to be 3,800,000 in 1948; furthermore, they say the rate of alcoholism in males and females increased 31 per cent and 33 per cent respectively from 1940 to 1948. This seems a large increase and even if it were only one-half that value, it can be seen why this is high on the list of socio-economic problems today. It is also high on the list of medical problems because it is often the family physician who first sees these problems.

It is with this background of information that the etiology of alcoholism is to be discussed. Organic factors will be emphasized, not because they are any more important than the psychological, but they lend themselves more easily to study; and if any of the hypotheses are proved to be true, they would lend themselves more readily to therapy.

These theories have fallen into three groups: (1) endocrine malfunction, (2) nutritional deficiency, and (3) allergic reaction. They will be discussed in that order.

ENDOCRINE MALFUNCTION

Although alcoholism has long been a problem—at least drunkenness has long plagued mankind—the reasons for a person losing control over the impulse to drink excessively were thought of as being moralistic in nature until relatively recently in the history of our civilization. Apparently a psychological etiology was put forth earlier than organic or physiological reasons.

Freud, in the early 1900's, and Bleuher, about 1910, discussed the psychodynamics of alcoholism.[4] It was not until 1925 that there was much discussion of a physiological cause of alcoholism. M. Gegon and H. Oclenmatt[5] at that time wrote a paper which dealt with dietary deficiency and chronic alcoholism. Gross[5] states this was the first paper appearing in the literature which mentioned possible organic factors as inducing alcoholism.

Gross, in a paper published in 1945 but actually written some 6 years earlier, discusses briefly the concept that alcoholism might have a physiological basis, but he thought it much more likely that the symptoms we note in chronic alcoholics were secondary to alcoholic toxicity on the endocrine glands, particularly the pituitary gland. He had noted definite histological changes in the cells of the pituitary gland and would attribute much of the somatic change seen in chronic alcoholics to changes in the endocrine functioning of that gland. It will be seen that this is the beginning of a concept which says that endocrine changes come secondarily to alcoholic ingestion, in contrast to a hypothesis discussed later which places endocrine changes as primary and predisposing to alcoholism.

Smith, in 1949,[6] hypothesized that alcoholism was a physiological manifestation of an adrenal insufficiency. He came to this conclusion after making repeated biochemical and clinical observations of patients in a hypoadrenal state and those in delirium tremens. He compared the behavior changes seen in other organic metabolic diseases such as hyperthy-

---

This is one of 11 theses, written by fourth year students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group judged to be the best by the faculty at the school.

Thesis written while the author was a fourth year student. Dr. Parman is now interning at General Hospital, Kansas City, Missouri.

almost this quick...

filmtab* Erythrocin®

starts to dissolve

NEW

filmtab* ...for faster drug absorption

Now, *there's no delayed action from an enteric coating.* The new tissue-thin *Filmtab* coating (marketed only by Abbott) starts to disintegrate within 30 seconds after your patient swallows it—makes the antibiotic available for immediate absorption.

NEW

filmtab* ...for earlier blood levels

Because of the swift absorption, your patient gets high blood levels of ERYTHROCIN (Erythromycin Stearate, Abbott) in *less than 2 hours*—instead of 4-6 hours as before. Peak concentration is reached within 4 hours, with significant concentrations lasting for 8 hours.

NEW

filmtab* ...for your patients

It's easy on them. Compared with most other widely-used antibiotics, *Filmtab* ERYTHROCIN is *less likely to alter normal intestinal flora.* Prescribe *Filmtab* ERYTHROCIN for all susceptible coccic infections—especially when the organism is resistant to other antibiotics. Bottles of 25 and 100 (100 and 200 mg.).

Abbott

**TM for Abbott's film sealed tablets, pat. applied for*

408174

roidism, menopausal syndrome, and cerebral arteriosclerosis.

Smith[6] feels that when one views the end result of delirium tremens and an Addison's crisis, one cannot keep from noting the similarity. In both, the plasma potassium and nitrogen are elevated and the sodium and chlorides are decreased. In both there is hemoconcentration, sometimes acidosis, and sometimes low blood sugar. Clinically, both states are characterized by malaise, nausea, vomiting, slow pulse, low blood pressure, and terminally in both there is a thready pulse with hyperpyrexia and extreme tachypnea.

According to Smith the treatment is the same—the administration of sodium chloride and glucose. Smith says that the biochemical, clinical, and therapeutic aspects of these two disease states are so similar that the existence of identical pathologic processes must be presumed.

Smith futhermore said that the clinical pictures of delirium tremens, Addison's crisis, and pantothenic acid deficiency resembled each other and this was another possibility which should be investigated. He also noted in acute intoxication a lymphocytosis, decreased plasma ascorbic acid, and some five other biochemical changes which, to him, indicated there was a metabolic basis for alcoholism. He also felt that he noted a definite somatic body type which was associated with a higher than normal incidence of alcoholism. This concept and a clinical trial of cortisone and ACTH were reported upon more completely by Tintera and Lowell[7] in 1949.

In 1949, Tintera and Lowell[7] reported in a paper on the treatment of alcoholism that there are two classes into which most alcoholics fall. The first class is made up of those individuals who are more likely to develop alcohol addiction when young. These are characterized as being young males who have asthenic habits, soft smooth faces, little or no chest hair, are hypotensive, and not infrequently have gynecomastia. This clinical picture, the authors thought, is compatible with a picture of hypoadrenal corticalism and is often seen in patients with a so-called allergic diathesis. These individuals are supposed to have hypoglycemia, low 17 ketosteroids, and low androgens.

The second class is the older group who develop a hypoadrenal state secondary to excessive alcohol ingestions. It had been reported earlier that there was a decrease of lipoid material in the adrenal cortices on chronic alcoholics coming to autopsy.[1]

These authors noted a low blood sugar (ranging from 54 to 80 mg. per cent) in a series of 200 patients they studied and concluded that the low blood sugar induced a craving for carbohydrate ingestion. Had the patient been anything more than a moderate drinker, he might develop alcohol addiction. They furthermore concluded from clinical and laboratory observation, together with satisfactory therapeutic results from giving ACTH and cortisone, that it seemed reasonable to assume that alcoholics were in a state of frank or latent hypoadrenalism.

Later observations of Mann[8] on a smaller group of alcoholics (95 in this group and 200 in Tintera and Lowell's group) failed to confirm the hypoadrenal state noted by the above workers. Mann used the Thorne test and found that four hours after 0.3 mg. of epinephrine was given subcutaneously, 62 per cent gave a normal response, a drop of 50 per cent or more in eosinophil count. A slightly subnormal response, a drop of 40 to 50 per cent, was noted in 26 per cent, while 12 per cent gave a decrease of less than 40 per cent in the eosinophil count. They doubt the validity of previous observations of hypoadrenalism in alcoholics, either frank or latent.

RELATIVE NUTRITIONAL DEFICIENCY

Several years ago some South American scientists[9,10] reported the presence of a thermolabile factor in brewer's yeast which, when absent from the diet of experimental animals, would cause these animals under autoselective methods to increase their alcohol consumption. This substance they called "Factor N." They therefore hypothesized that they had found a new member of the B-Complex group and even established a unit of this factor based on its abilities to reduce alcohol consumption. This they thought might serve as a useful therapeutic aid in preventing and treating alcoholism. No satisfactory follow-up on their work could be found.

In 1947, Williams,[11,12] after reviewing the work of the above authors and the work of Brady and Westerfield,[13] made a hypothesis that alcoholism was a "genetotrophic" disease. He postulated that there is genetic individuality for every person and, because of such predetermined genetic factors in people, there was likewise a biochemical individuality. Developing this hypothesis further, he thought that certain individuals required more of certain foodstuffs than others, especially vitamins and minerals. Hence, he concluded that some people might actually be on a nutritionally deficient diet while others on the same diet might be adequately nourished. He next concluded that when diet is inadequate there develop cravings for food, and with some people there develops an intense craving for alcohol. This craving he compared to the craving for sodium chloride in an adrenalectomized animal.

In order to prove or disprove his hypothesis, he began a series of experiments with rats. With these animals he felt he could establish individual metabolic needs, and when these needs were not fulfilled the rats would drink more alcohol under autoselective methods. The next step was to add to the deficient diets a highly nutrient supplement containing large amounts of all of the recognized vitamins

UNIVERSITY OF KANSAS SCHOOL OF MEDICINE

POSTGRADUATE MEDICAL COURSE

OBSTETRICS

November 8, 9 and 10, 1954

Guest Instructors:

JOHN I. BREWER, M.D., Professor of Obstetrics & Gynecology, Northwestern University Medical School, Chicago, Ill.

F. BAYARD CARTER, M.D., Professor of Obstetrics & Gynecology and Head of Department, Duke University School of Medicine, Durham, N. C.

CARL P. HUBER, M.D., Professor of Obstetrics & Gynecology and Chairman of Department, Indiana University School of Medicine, Indianapolis.

ROBERT A. KIMBROUGH, JR., M.D., Professor of Obstetrics & Gynecology and Chairman of Department, University of Pennsylvania Postgraduate School of Medicine, Philadelphia.

CURTIS J. LUND, M.D., Professor of Obstetrics & Gynecology and Chief of Department, University of Rochester School of Medicine, Rochester, N. Y.

WILLIAM F. MENGERT, M.D., Professor of Obstetrics & Gynecology and Chairman of Department, Southwestern Medical School, University of Texas, Dallas.

Subjects to Be Discussed:

Fibroids and Pregnancy

FIBROIDS AND STERILITY: Do Fibroids Produce Infertility; When and How; Place of Myomectomy in Treating Infertility.
FIBROIDS AND ABORTION: Can One Predict That an Abortion Will Result; After One (Two or Three) Abortion, What Do We Do?
COURSE OF PREGNANCY: The Large Fibroid; Multiple Subserous Fibroids; The "Large" Uterus at Two or Three Months.
ACUTE DEGENERATION: Medical or Surgical; Timing of Operation; Prognosis for Continuation of Pregnancy.
USUAL TYPES OF FIBROIDS: (Obstructing and Non-Obstructing)—The Pediculated Fibroid in Vagina; The Pediculated Fibroid in Cul-de-sac.
COURSE OF LABOR: First Stage; Third Stage.
THE PUERPERIUM AND THE FUTURE: Degree of Expected Involution; When Myomectomy; When Hysterectomy?

Placenta Previa

ETIOLOGY: Is There an Etiology or Is It Merely a Matter of Chance?
DIFFERENTIAL DIAGNOSIS, INCLUDING DEGREE OF PREVIA.
MEDICAL TREATMENT.
SURGICAL TREATMENT.
THIRD STAGE AND NEXT FEW HOURS.
PROGNOSIS FOR CHILD AND FOR NEXT PREGNANCY.

Premature Separation

ETIOLOGY: Role of Toxemia; Venous Pressure.
ROLE OF MEDICAL TREATMENT: Minor Degrees of Separation; Twin Pregnancy; Major Separation.
SURGICAL TREATMENT: When? What?
PROGNOSIS: Baby Now Alive; Future Pregnancies.

The Official Meeting of the Kansas State Obstetrical Society



Woodcroft Hospital--Pueblo, Colorado

A private hospital for the scientific treatment of neuro-psychiatric disorders, including alcoholism and drug addiction. Beautiful landscaping and home-like surroundings afford a restful atmosphere. Accommodations vary from single rooms with or without bath to rooms en suite, allowing for segregation of guests.

Detailed information furnished on request.

KARL J. WAGGENER, M.D.

and essential minerals. He noted that rats predisposed to alcohol ingestion decreased their intake of alcohol under this regimen.

The final step was to give his idea a clinical trial. This he did in 1950 on a group of 20 patients. In 1951[14] he published a small book in which he states that the early results appeared good but were not conclusive. The results seemed good in spite of the great difficulties he had in working with alcoholics who would follow this regimen poorly.

Since the 1951 preliminary report, no follow-up could be found except indirectly through Smith,[15] who reported on six of the 20 patients. He states, after a period of 7 to 10 months, that two showed some improvement, one could now drink moderately, and one could tolerate larger quantities of alcohol without showing toxic signs. Smith at that time could add nothing new on an organic cause of alcoholism.

Sirnes, in 1953, noted that rats in which he induced hepatic fibrosis by using carbon tetrachloride showed an increased alcohol ingestion by autoselective methods. The increase was up to four times that of the normal rat. While this does not give a direct clue to the etiology of alcoholism, it might explain why, if hepatic fibrosis were induced by other than malnutrition of alcoholism, there was an increase of alcohol consumption.

Lester and Greenberg,[17] in a recently published paper, re-evaluated the work of Williams and did a series of experiments with rats under autoselective methods. The scope of their work was broader than that of Williams as they gave the animals a wider choice of fluids to drink and added carbohydrate supplements to the diet. They noted that the animals would choose a sucrose solution in preference to a 7.6 per cent alcohol solution when placed on caloric deficient diets. This preference was, according to them, independent of the vitamins in the diet and had as its basis a caloric insufficiency. They hypothesized that the reason the animals in Williams' experiments consumed more alcohol was to increase their caloric intake.

Westerfield and Lawrow[18] more recently noted the work of Lester and Greenberg and then studied the effect on alcohol consumption of a calorie and thiamine deficient diet versus a solely calorie deficient diet. They noted that thiamine deficient diets did reduce food intake, but in their work they did not find the expected increase of alcohol consumption accompanying it as Mardones had indicated would happen. Secondly, they noted that the actual alcohol consumption was greater in the group of animals on the plain calorie deficient diet than in that group on the vitamin deficient-calorie deficient diet, as might be expected from work by Williams and Mardones and earlier work of their own. Thus, it can be seen that the results of the experiments of a nutritional concept of alcoholism are far from conclusive.

ALLERGY

Lester and Greenberg[19] published a paper in 1951 in which they briefly discussed allergy as a factor in producing alcohol addiction. They say that allergic theories of alcohol addiction were advanced many years ago, but some work of Robinson and Voeglten[21] conclusively demonstates that ethyl alcohol per se is not an allergen. They note as at least a theoretical possibility that the side products present in the beverage—whether they be rye, grape, corn, or whatever the source—might serve as allergens. They go on to say that there has been no convincing proof offered to support such an allergic theory of alcohol addiction and that no mention of an immunological basis of alcohol addiction could be found elsewhere.

DISCUSSION

Wexberg,[22] in surveying the physiopathological theories of the etiology of alcoholism, points out some interesting facts with reference to the work of Smith and Williams. Wexberg believes that the adrenal insufficiency of Addison's disease and the clinical state of delirium tremens are probably quite similar if not the same; but he doubts seriously if it can be said that alcoholism is primarily a metabolic disease with a secondarily manifested behavior change. He points out that most if not all of Smith's patients were already alcoholics.

To support this view, Wexberg quotes the extensive review and study made by Goldfarb and Berman[23] wherein they found multiple endocrine changes secondary to chronic alcoholism. Inasmuch as most of Smith's patients were already alcoholics, he doubts if the criteria he used to judge the premorbid type were valid, especially with reference to the younger group. He does not doubt that alcohol acts as a stressful agent and that there are secondary endocrine changes not only in the adrenal but in the pituitary and elsewhere in the endocrine system as reported by Goldfarb and Berman.

With reference to the work of Williams, he believes that the craving for sodium chloride following adrenalectomy and the craving for alcohol are far from being comparable. The craving for sodium chloride is for a product which is naturally a part of our body tissues, whereas alcohol is not. He believes that before any physiopathological theory can be considered adequate to explain the etiology of alcoholism, there should be an adequate number of people possessing the premorbid type, whether it be endocrine or metabolic, who did develop alcoholism and an insignificant number of people with the premorbid type who did not develop alcoholism. Wexberg feels this was not the case with any of the workers above who proposed theories of a physiopathological etiology of alcoholism. Clinically, they do not deny that ACTH and cortisone will help in

It's Convenient to Buy Kanox

THERAPEUTIC OXYGEN AND ANESTHETIC GASES



Oxygen Therapy
Regulators and Equipment
Repair Service for
Regulators, Flowmeters
and Humidifiers

Nitrous Oxide
Ethylene
Carbon Dioxide
Helium
Oxygen
Cyclopropane

KANSAS OXYGEN, Inc.

Phone 5-5551 HUTCHINSON, KANSAS Box 551

Pure as sunlight



the treatment of alcoholism, but they believe the improvement is because of a secondary and not a primary change in the adrenals.

More recently Paphan[24] wrote a critique of Williams' work, emphasized that it would be a tedious task to establish the individual metabolic needs of many patients, and doubts that Williams conducted such a thorough investigation. This he feels does not completely invalidate Williams' work but should serve as a stimulus for more complete investigation.

In view of a not too clearly defined physiological cause of alcoholism, it would seem fitting and proper to consider the other most currently popular etiological cause, a psychological etiology. As mentioned before, Freud and Bleuher early discussed the psychodynamics of alcoholism. Since then there have been numerous papers by psychologists and psychiatrists,[25,26,27,28] depicting a certain type of personality which is predisposed to alcoholism.

Some authors have emphasized the deep masochistic nature of alcoholics,[25] others latent or overt homosexuality, and others just say they are unhappy individuals who fail to adjust well or stand stress well.[31] In any event, some of the most recent studies and reviews of the many papers on personality types fail to find any specific prealcoholic type.[29,30]

In a more superficial manner, one can say the alcoholic is better able to face reality when he is drinking. This he may do by completely misjudging reality as it is, or he may find that alcohol gives him the extra boost his ego needs to perform the task necessary to place his psyche in a state of homeostasis.

The psychodynamics of alcoholism today are most easily understood when based on the Freudian concept of psychosexual development. It is postulated that because of numerous psychic traumata, the individual is unable to stand the psychic stresses of life. Because of this instability of personality structure, the patient regresses to a more infantile level of psychosexual development, whether it be to the oral dependent stage or to some other stage of development. In any event, the person meets with reality by drinking and, according to Lewis,[31] in preference to actually becoming psychotic, the individual takes a more socially acceptable means of facing reality—that of drinking alcohol.

According to Knight, the stress or tension which is being relieved is induced by frustrated passive needs which were engendered by parents. When the affected individual finds that alcohol will relieve tension, he passes that way more and in every increasing frequency until his perspective of reality is so distorted that he cannot adequately deal with reality.

Higgins,[33] in a recent review of the psychodynamics of alcoholism, believes essentially what has been outlined above. However, he places less emphasis on the oral dependent nature of the alcoholic. He believes that the regression can be to any stage of development, and the tension is not always the result of frustrated passive needs but may be due to adult sexual conflicts. He believes that the depressant action of alcohol is such as to facilitate the operation of a normal defense mechanism which previously was unable to function in its proper role. Higgins developed this modification of the more commonly accepted oral dependence concept after studying many alcoholics who were being treated as office patients with Antabuse. He felt his picture of the personality disorder was different and more accurate than that obtained by earlier workers because most of his patients had been alcoholics for a relatively short time.

CONCLUSION

In reviewing the available literature, a good physiological etiology for alcoholism could not be found. At the onset, the work of Williams looked good, but it did have some theoretical pitfalls. Also, later workers were not able to confirm his concepts in the exact manner he presented them. It may be, as he pointed out in his original paper, that inasmuch as alcoholics are difficult to work with, several years may pass before a good clinical study of his theories could be finished. In spite of disappointing early reports, this concept deserves more thought.

The psychological theories are not specific and do not give any clear reasons why some people actually stand stress poorly. They say there has been a fixation at some stage in their psychosexual development. Why it is that these individuals cannot grow into mature personalities is not explained.

A good single etiological cause for alcoholism could not be found among the physiological theories which have been advanced. Maybe the relative nutritional deficient states and endocrine malfunctioning lead to poor stress endurance which we interpret as psychic instability. Perhaps this is another condition in which multiple etiological factors are involved.

Nationally advertised Surgical Supplies and Equipment have been placed at Topeka, Joplin, Kansas City and St. Joseph for your convenience by—

GOETZE NIEMER COMPANY

Management by Dr. W. F. Goetze, member of the American Medical Ass'n, assures intelligent servicing of your orders.

Medical and Surgical Supplies for Doctors of Medicine and Hospitals

Munns Medical Supply Co.
512 Kansas Avenue
Topeka, Kansas

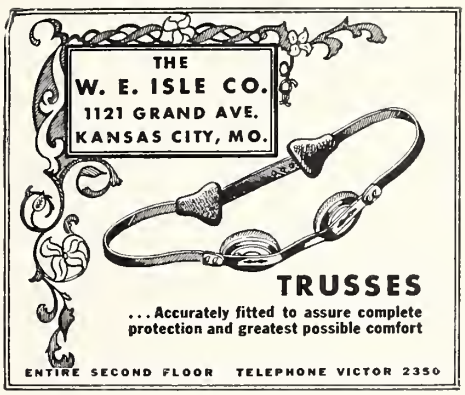
CLASSIFIED ADVERTISEMENTS

FOR SALE—Modern office equipment of deceased physician. Used only four years. Equipment for general practice, including x-ray. Write the Journal 15-54.

WANTED: Dermatologist for seven-man group in Midwestern city. Above average facilities with excellent financial opportunity. Write the Journal 13-54.

FOR SALE—Office equipment, Picker Duo-column portable x-ray, diathermy, lamps, instruments, medical books and other equipment. Very reasonable prices. Write the Journal 14-54.

WANTED: Pediatrician for seven-man group in Midwestern city. Above average facilities with excellent financial opportunity. Write the Journal 12-54.



MUTUAL TRUST

Offered by

MUTUAL DISTRIBUTORS, Inc.
1016 Baltimore • Kansas City 5, Mo.

Principal Underwriter and Distributor

PROSPECTUS ON REQUEST

PLEASE SEND ME PROSPECTUS

*Name*_____

*Address*_____

*City*_____ *State*_____

Clarence W. Glassen, State Representative

THE LATTIMORE-FINK LABORATORIES

Topeka — El Dorado
Kansas

J. L. Lattimore, A.B., M.D., Pathologist
A. A. Fink, A.B., M.D., Pathologist
H. C. Ebendorf, M.T., Serologist
A. C. Keith, B.S., Chemist
L. W. Hull, A.B., Bacteriologist
Walter Norris, A.B., Chemist

Anatomical and Clinical Pathology

Containers Furnished Upon Request

Careful laboratory and clinical investigation will solve this big problem of alcoholism in time.

BIBLIOGRAPHY

1. Krantz, J. C., and Carr, C. J.: Pharmacologic principles of medical practice, ed. 2, Baltimore, Williams and Wilkins, p. 531, 1951.
2. Smillie, Wilson G.: Preventive medicine and public health, ed. 2, New York, Macmillan, p. 438, 1952.
3. Jellinek, E. M., and Keller, Mark: Rates of alcoholism in the United States 1940-48, Quarterly Journal of Studies on Alcohol 13:49-59 (Mar.) 1952.
4. Lorand, Sandor: A survey of psychoanalytic literature on problems of alcohol: bibliography, The Year Book of Psychoanalysis, New York, International Universities Press, 1945, vol. 1, 359-70.
5. Gross, M.: The relation of the pituitary gland to some of the symptoms of alcohol intoxication, Q.J.S.A. 6:25-35 (Mar.) 1945.
6. Smith, J. J.: Alcoholism from a medical viewpoint, Q.J.S.A. 10:251-57 (June) 1949.
7. Tintera, John W., and Lowell, Harold W.: Endocrine treatment of alcoholism, Geriatrics 4:274-80 (Sept. and Oct.) 1949.
8. Mann, Norman M.: Hypoadrenalism and alcoholism, Q.J.S.A. 13:201-03 (June) 1952.
9. Mardones, R. J.: On relationship between deficiency of B vitamins and alcohol intake of rats, Q.J.S.A. 12:563-75 (Dec.) 1951.
10. Mardones, R. J.; Sergonia, N., and Onfray, E.: Relationship between the dose of factor N and alcohol intake of rats under autoselective conditions, 9:401-06 (Mar.) 1946.
11. Williams, R. J.: The etiology of alcoholism: A working hypothesis involving the interplay of heredity and environmental factors, Q.J.S.A. 7:567-87 (Dec.) 1946.
12. Williams, R. J.; Berry, L. J., and Beerstecher, E.: Biochemical individuality, Archives of Biochemistry 23:275-90 (Sept.) 1949.
13. Westerfield, W. W., and Brady, R. A.: The effect of B-complex vitamins on voluntary consumption of alcohol, Q.J.S.A. 7:499-507 (Dec.) 1946.
14. Williams, R. J.: Nutrition and Alcoholism, Norman, Okla., University of Oklahoma Press, 1951.
15. Smith, J. A.: Alcoholism: Causes and methods of treatment, Ameriean Practitioner and Digest of Treatment 4:1-21 (July) 1953.
16. Sirnes, Tollok B.: Voluntary consumption of alcohol in rats with cirrhosis of the liver, Q.J.S.A. 14:3-18 (Mar.) 1953.
17. Lester, David, and Greenberg, Leon: The effect of sucrose, fat, and saccharin on self selection of alcohol by rats, Q.J.S.A. 13:553-60 (Dec.) 1952.
18. Westerfield, W. W., and Lawrow, John W.: The effect of caloric restriction and thiamine deficiency on voluntary consumption of alcohol in rats, Q.J.S.A. 14:378-84 (Sept.) 1953.
19. Lester, David: Alcoholism 1941-51: A survey of research, education, and therapy, Q.J.S.A. 13:444-52 (Sept.) 1952.
20. Voeglten, Walter L., and Robinson, M. W.: The role of allergy in alcoholism, Q.J.S.A. 13:196-200 (June) 1952.
21. Wexberg, Leopold E.: A critique of the physiological theories of alcoholism, Q.J.S.A. 11:113-118 (Mar.) 1950.
22. Goldfarb, Alvin, and Berman, Sidney: Alcoholism, a psychosomatic disorder, Q.J.S.A. 10:415-29 (Sept.) 1949.
23. Paphan, R. E.: A critique of the genetotrophic theory of the etiology of alcoholism, Q.J.S.A. 14:228-37 (June) 1953.
24. Moras, Nathan: Alcoholic personality, Q.J.S.A. 3:45-50 (Mar.) 1942.
25. Bergler, Edmund: Personality traits of alcohol addicts, Q.J.S.A. 7:356-59 (Dec.) 1946.
26. Lolli, Giorigio: The addictive drinker, Q.J.S.A. 10:405-14 (Dec.) 1949.
27. Lolli, Giorigio: Treatment of alcohol addiction, Q.J.S.A. 13:461-71 (Sept.) 1952.
28. Botwinic, J., and Machover, Sellomon: Personality traits in alcoholics: A eritique of existing studies, Q.J.S.A. 12:268-72 (June) 1951.
29. Sutherland, E. H.; Schrolder, H. G., and Tordella, L.: Personality traits and the alcoholic: A critique of existing studies, Q.J.S.A. 11:547-61 (Dec.) 1950.
30. Lewis, N. D.: Personality factors in alcohol addiction. Q.J.S.A. 1:21-44 (Mar.) 1940.
31. Ullman, A. D.: The psychological mechanism of alcohol addiction, Q.J.S.A. 13:602-08 (Dec.) 1952.
32. Higgins, John W.: Psychodynamics of alcoholism, A.M.A. Archives of Neurology and Psychiatry 69:713-31 (June) 1953.
33. Vogel, Sidney: Dynamics of alcoholism, Q.J.S.A. 14:620-31 (Dec.) 1953.

Medical Board Elects

Dr. J. D. Colt, Jr., Manhattan, was re-elected president of the Kansas State Board of Medical Registration and Examination at a meeting held in Kansas City in June. Dr. H. E. Haskins, Kingman, was named to serve again as vice-president. The secretary, Dr. O. W. Davidson, Kansas City, is serving a four-year term.

At the time of the meeting, 116 persons took examinations for licenses to practice medicine and surgery in Kansas. Of that number, 103 are graduates of the University of Kansas School of Medicine.

Southeast Kansas Society Meets

The Southeast Kansas Medical Society met at the Tioga Hotel, Chanute, on June 9. Dr. Dick B. McKee, Pittsburg, introduced four physicians from the Wichita Clinic who presented a symposium on "Low Back Pain." Participants were Dr. John F. Lance, Dr. N. C. Siebert, Dr. John W. Warren, and Dr. Arthur H. Bacon.

A national inventory of nursing homes and related facilities is being conducted by the Division of Hospital Facilities, Public Health Service. The inventory will secure information for providing better criteria for developing state programs for the care of the chronically ill by taking into account the role and extent of nursing homes in relation to the need for chronic disease hospital facilities.

RENTALS — SALES

HOME PATIENT AIDS
• Bed Pans
• Urinals
• Crutches
• Wheel Chairs
• Invalid Walkers

SICK ROOM EQUIPMENT
• Hospital Beds Complete
• Bedside Commodes
• Bedside Tables
• Invalid Lifts
• Heat Lamps

Many Other Items

★ PETRO'S RENTAL SERVICE ★

618-20 Quincy — Topeka, Kansas — Phone 40207

Current reports[1,2] describe the increasing incidence of resistance among many pathogenic strains of microorganisms to some of the antibiotics commonly in use. Because this phenomenon is often less marked following administration of CHLOROMYCETIN (chloramphenicol, Parke-Davis), this notably effective, broad spectrum antibiotic is frequently effective where other antibiotics fail.

Chloromycetin®

Coliform bacilli—100 strains

up to 43% resistant to other antibiotics;
2% resistant to CHLOROMYCETIN.[1]

Staphylococcus aureus—500 strains

up to 73% resistant to other antibiotics;
2.4% resistant to CHLOROMYCETIN.[2]

CHLOROMYCETIN is a potent therapeutic agent and, because certain blood dyscrasias have been associated with its administration, it should not be used indiscriminately or for minor infections. Furthermore, as with certain other drugs, adequate blood studies should be made when the patient requires prolonged or intermittent therapy.

References
(1) Kirby, W. M. M.; Waddington, W. S., & Doornink, G. M.: Antibiotics Annual, 1953-1954, New York, Medical Encyclopedia, Inc., 1953, p. 285.
(2) Finland, M., & Haight, T. H.: *Arch. Int. Med.* **91**:143, 1953.

TABLE OF CONTENTS

SEPTEMBER, 1954

ORIGINAL ARTICLES

Lung Cysts—Melvin A. Rabe, M.D., Leavenworth, Kansas 489

Immunity and Resistance in Poliomyelitis—Herbert A. Wenner, M.D., Kansas City, Kansas . 495

Rupture of the Colon Secondary to Muscular Strain —W. G. Cauble, M.D., Wichita, Kansas . 500

An Error in Blood Sugar Determinations—Mark Dodge, M.D., Marion Mathy, Arthur W. Robinson, M.D., and Arnold V. Arms, M.D., Kansas City, Missouri 502

EDITORIALS

A History of the Society 507
Federal Health Programs 507
A.M.A. Annual Session 508

MISCELLANEOUS

President's Page 506
Clinicopathological Conference 519
Adverse Effects of Penicillin Therapy—Senior Thesis 536

Entered as second-class matter at the post office at Fulton, Missouri, June 19, 1953, under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of October 3, 1917, authorized on July 2, 1918. Office of Publication: 1201-05 Bluff Street, Fulton, Missouri.

THE JOURNAL OF THE KANSAS MEDICAL SOCIETY

Editorial Board

ORVILLE R. CLARK, M.D., *Editor,* Topeka

JOHN W. CAVANAUGH, M.D., Topeka

DAVID E. GRAY, M.D., Topeka

RICHARD GREER, M.D., Topeka

DWIGHT LAWSON, M.D., Topeka

Associate Editors

DONALD P. TREES, M.D., Wichita

GLEN R. SHEPHERD, M.D., Kansas City

Information

Ownership: The Journal is a non-profit publication owned and published monthly by the Kansas Medical Society.

Subscription: A year's subscription to the Journal is included in membership in the Kansas Medical Society, with $2.00 of each member's dues apportioned to the Journal. Rates to others, except in foreign countries, $4.00 per year or 60c per copy.

Material: Scientific articles, editorials, and data of general interest are invited from all members. Articles are to be submitted on condition that they are contributed solely to this publication. A right is reserved to reject any material deemed unsatisfactory.

Manuscripts: Only manuscripts that are typewritten on one side, double spaced, and original copies can be accepted. Manuscripts will be returned upon request.

Advertising: All advertising contracts, and all copy from advertisers under contract are subject to approval of the editorial board. Copy should be received by the 20th of the month immediately preceding the month of publication.

Non-Responsibility: Although an effort is made to publish only accurate articles and legitimate advertisements. the Journal denies legal responsibility for any statements, opinions, or advertisements appearing under the names of contributors or concerns.

PAULINE FARRELL, Managing Editor
and Advertising Manager

OLIVER E. EBEL, Business Manager

THE KANSAS MEDICAL SOCIETY

President . . . . . MURRAY C. EDDY, M.D., Hays
Past President . . . . . LUCIEN R. PYLE, M.D., Topeka
President-Elect . . . . . JOHN M. PORTER, M.D., Concordia
First Vice-President . . . CLYDE W. MILLER, M.D., Wichita
Second Vice-President, CONRAD M. BARNES, M.D., Seneca
Constitutional Secretary, JAMES A. BUTIN, M.D., Chanute
Treasurer . . . . . JOHN L. LATTIMORE, M.D., Topeka
A.M.A. Delegate, 1954-1955, GEORGE F. GSELL, M.D., Wichita
A.M.A. Delegate, 1955-1956, LAURENCE S. NELSON, M.D., Salina

Councilors and Councilor Districts

First District, FREDERICK E. WRIGHTMAN, M.D., Sabetha
Second District . . . GLENN R. PETERS, M.D., Kansas City
Third District . . . . . H. PENFIELD JONES, M.D., Lawrence
Fourth District . . . . CHARLES E. VESTLE, M.D., Humboldt
Fifth District, SEVERT A. ANDERSON, M.D., Clay Center
Sixth District . . . . . FLOYD C. TAGGART, M.D., Topeka
Seventh District . . . . . TO BE ANNOUNCED
Eighth District . . . . . JAMES E. HILL, M.D., Arkansas City
Ninth District . . . . LAURENCE S. NELSON, JR., M.D., Salina
Tenth District . . . . . HAROLD M. GLOVER, M.D., Newton
Eleventh District . . . . NORTON L. FRANCIS, M.D., Wichita
Twelfth District . . . . . CYRIL V. BLACK, M.D., Pratt
Thirteenth District . . LLOYD W. REYNOLDS, M.D., Hays
Fourteenth District . . . . JUSTIN A. BLOUNT, M.D., Larned
Fifteenth District . . . . ROBERT G. KLEIN, M.D., Dodge City
Sixteenth District . . . . . JAMES L. JENSON, M.D., Colby
Seventeenth District . . . . . H. PRESTON PALMER, M.D., Scott City

OLIVER E. EBEL, EXECUTIVE SECRETARY
RUEBEN M. DALBEC, EXECUTIVE ASSISTANT

Executive Office: 315 West Fourth Street, Topeka

THE JOURNAL
of the
KANSAS MEDICAL SOCIETY

Copyright, 1954, by The Kansas Medical Society

Volume LV SEPTEMBER, 1954 No. 9

Lung Cysts

Melvin A. Rabe, M.D.*
Leavenworth, Kansas

Lung cysts are neither a new nor rare disease, but awareness of and interest in this disease entity has been comparatively recent. For the most part it has paralleled the development of thoracic surgery.

HISTORICAL BACKGROUND

Koontz[1] in 1925 reviewed the World's literature on cystic disease of the lung and found a total of 108 cases including allied conditions such as tracheal diverticula, cysts of aberrant lung tissue, etc. Most of these cases were reported by the Germans and at autopsy. There were *no* reports in the American literature at that time.

Eloesser[2] in 1931 could find but four reports of cystic disease of the lung in the American literature and added four cases of his own.

Wood[3] in 1934 reviewed twenty-three cases from the literature and added sixteen of his own. Interestingly, the treatment at that time seemed to be in doubt with only a few cases subjected to definitive surgical therapy.

Schenck[4] in 1937 stated that a total of only 381 cases of cystic disease of the lungs had been reported since the original description. This was several years after some of the monumental strides in chest surgery had already been made. The first successful pneumonectomy by Nissen[5] had been done and successful pneumonectomy for carcinoma had been carried out by Graham,[6] both in 1933. The modern technic of individual ligation of hilar structures had been developed by Rienhoff[7] later that same year, but thoracic surgery did not attain wide application for several years thereafter. The introduction of the technic of segmental resection by Churchill and Belsey[8] in 1939, together with increasing knowledge of thoracic physiology and anesthetic technics, set the stage for the surge of interest in chest surgery in World War II. Increasing awareness of chest diseases amenable to surgery, including cystic disease of the lung, is apparent in the literature from that time to the present. Indeed it has only been since World War II that reports of numerous cases of lung cysts have become fairly common in the literature.

PATHOLOGY

The nomenclature and pathogenesis of lung cysts still seems to be confused and controversial. Cooke and Blades[9] divide them into two types: congenital and acquired, considering only bullous emphysema and subpleural blebs as acquired lesions and dividing the congenital cysts into bronchiogenic and alveolar types.

Maier and Haight[10] classify them into two groups according to the presence or absence of an epithelial lining. They state that the epithelialized group may be congenital or acquired. The wall of a congenital epithelialized cyst may contain bronchial elements such as cartilage, glands, and smooth muscle. The acquired epithelialized cyst usually does not have cartilage and muscle in the wall; epithelialization results from an ingrowth of epithelium from the bronchial fistulas in the same manner as in an epithelialized, chronic, pulmonary abscess. The nonepithelial-

* Attending Surgeon, Veterans Administration Center, Wadsworth, Kansas.

One of the case reports in this article is from the Veterans Administration Center, Wadsworth, Kansas. The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

ized pulmonary cyst has a wall consisting merely of thin connective tissue without epithelium. There may be some flattened connective tissue cells in areas presenting a mesothelial lining, but there are no bronchial elements in the wall. Compressed alveoli surround the thin connective tissue lining. There is no definite line of distinction between these pulmonary cysts or pneumatoceles and bullous emphysema.

Moersch and Clagett[11] divide all pulmonary cysts into two chief types: those that originate from the bronchial tree and those that are alveolar in origin. Bronchiogenic cysts are characterized by the fact that they possess an epithelial lining which may or may not be ciliated. In contrast those cysts which are alveolar in type do not possess an epithelial lining but are lined with alveolar cells that are compressed together to form the wall.

There is also a variance of opinion regarding the etiology of these lesions. Practically all of the early writers seem to take for granted that these lesions were congenital. In contrast most of the present day workers, with the notable exception of Blades and his associates, seem to feel that many if not most of these lesions are acquired. This was first pointed out by Peirce and Dirkse[12] in 1937 who stated: "The common lack of information in regard to the antecedent respiratory history of patients with cystic pulmonary disease raises grave doubt of the propriety of the term 'congenital' in a roentgenologic diagnosis of cystic disease." They report a series of cases tending to substantiate a diagnosis of acquired cystic disease and quote Dr. Carl J. Weller, Director of the University of Michigan Pathology Laboratory, as stating that he has never seen changes in the lungs of infants or young children which would permit the diagnosis of congenital cystic disease of the lung. They conclude that a congenital origin for cystic pulmonary disease is doubted as a valid premise in a majority of cases. Ackerman[13] states that congenital cystic disease is often incorrectly diagnosed and the criteria for its recognition are poorly defined. He points out that lung abscesses may heal leaving a large unilateral cyst with a smooth lining and that alveolar cysts may form by the rupture of an alveolus into adjoining alveoli with the formation of a large air space by compression of the residual nondiseased lung as it enlarges. Thus both the so-called alveolar and bronchiogenic cysts may be acquired.

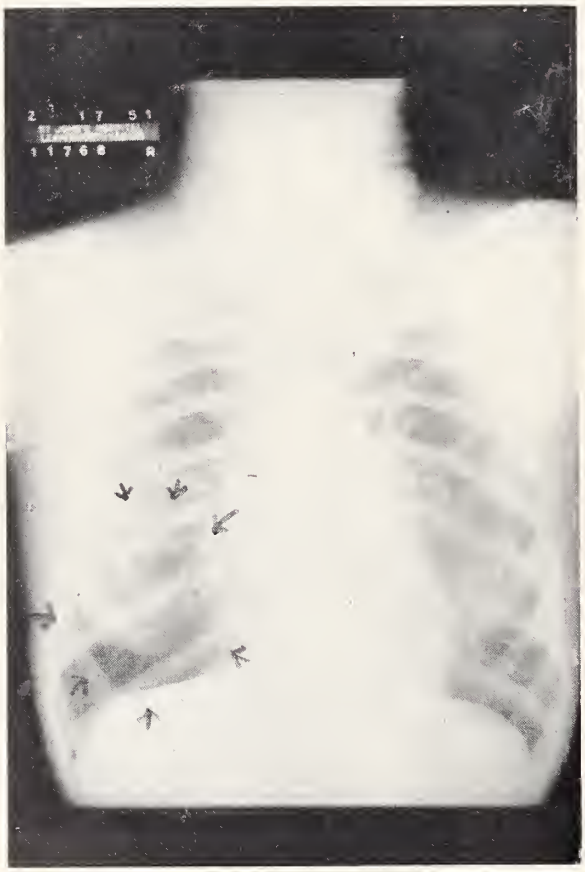


Fig. 1 (S. M.) 29 year old white female. Original film 2-17-51 in which a lung cyst at the base of the right lung was discovered. It is barely visible in the picture and is marked by arrows. It is more apparent on the film but could easily be missed by casual inspection.

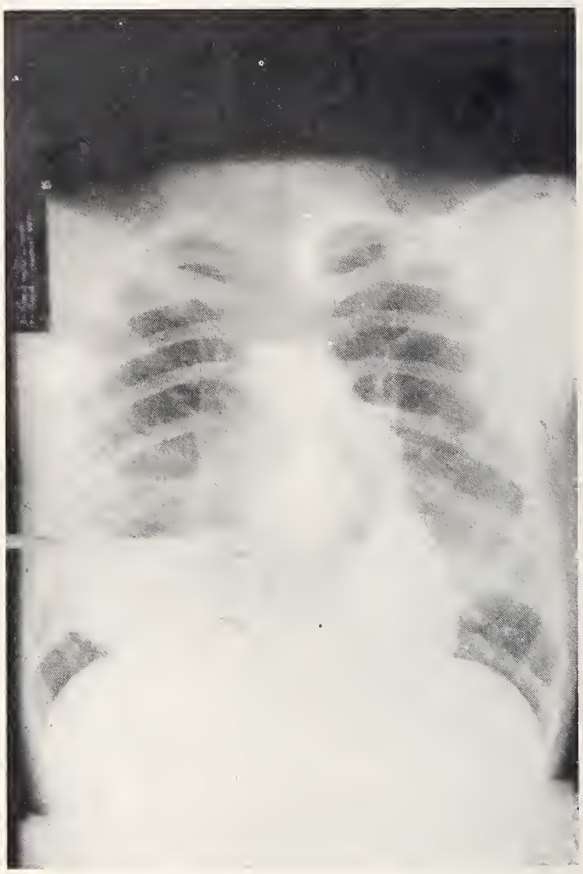


Fig. 2 (S. M.) Same patient 2 years later showing collection of fluid in cyst.

DIAGNOSIS AND TREATMENT

Moersch and Clagett in a review of 44 cases state that the symptomatology of the disease is dependent upon the status of the bronchial communication and the presence or absence of infection. In their series, cough, expectoration, hemoptysis, dyspnea, and pain were the most prevalent symptoms and in that order of frequency. Many cases are of course asymptomatic, especially those that are uninfected and those that have no bronchial communication. The incidence of relatively asymptomatic lung cysts is seen in a mass chest survey taken at Ft. Leavenworth, Kansas,[14] where four cases of lung cysts were found in a series of 3,435 routine chest x-rays of the population of the Post. The diagnosis is best made by means of chest x-rays which should include a lateral projection. Bronchography may occasionally be helpful, especially to differentiate cystic bronchiectasis. Bronchoscopy rarely gives much diagnostic aid.

The disease must be differentiated from pulmonary tuberculosis, pulmonary abscess, carcinoma, mediastinal tumors, empyema and pneumothorax. Fluid filled cysts have been mistakenly treated as empyema, and giant air cysts have been treated as spontaneous pneumothorax and subjected to aspiration of air.[15] When the diagnosis is in doubt, exploratory thoracotomy is advised by most authorities.

The treatment of the disease is entirely surgical and should be carried out even though the lesion is asymptomatic. This is to avoid the possibility of allowing more serious lesions to remain because the diagnosis cannot be positively made, as well as to prevent the development of carcinoma in the cyst, which was found in over 5 per cent of the cases reported by Moersch and Clagett. Then too, the asymptomatic cysts are prone to become symptomatic with the passage of time, especially if they have a bronchial communication. The surgical procedure advised by Dickson et al[16] is one which will extirpate the cyst with conservation of as much functioning lung as possible. This may be done by enucleation or wedge resection although occasionally lobectomy or rarely even pneumonectomy may be necessary. Allbritten and Templeton[17] recommend that single large cysts of the lung be treated by simple excision of the walls of the space, closure of bronchiolar fistulas, and obliteration of the pleural space by re-expansion of the lung with catheter drainage and suction.

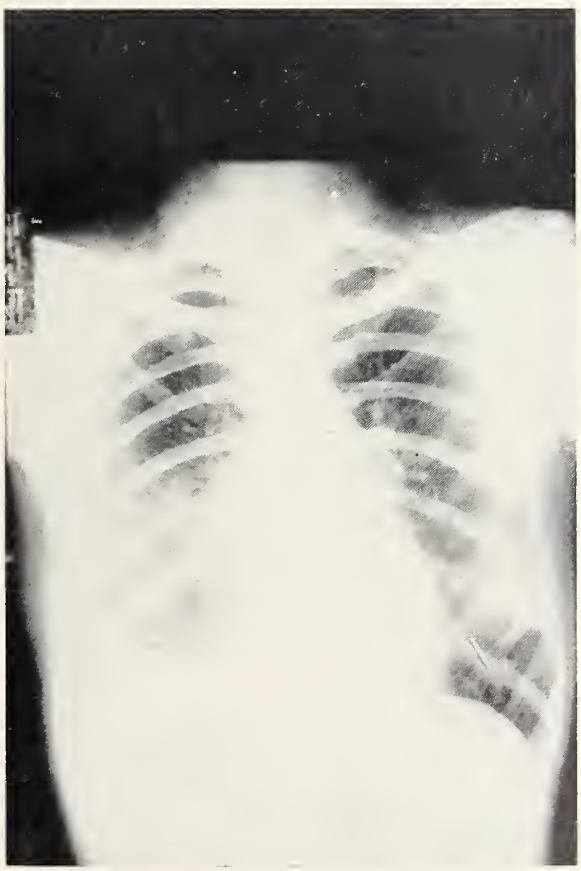


Fig. 3 (S. M.) Same patient postoperatively.

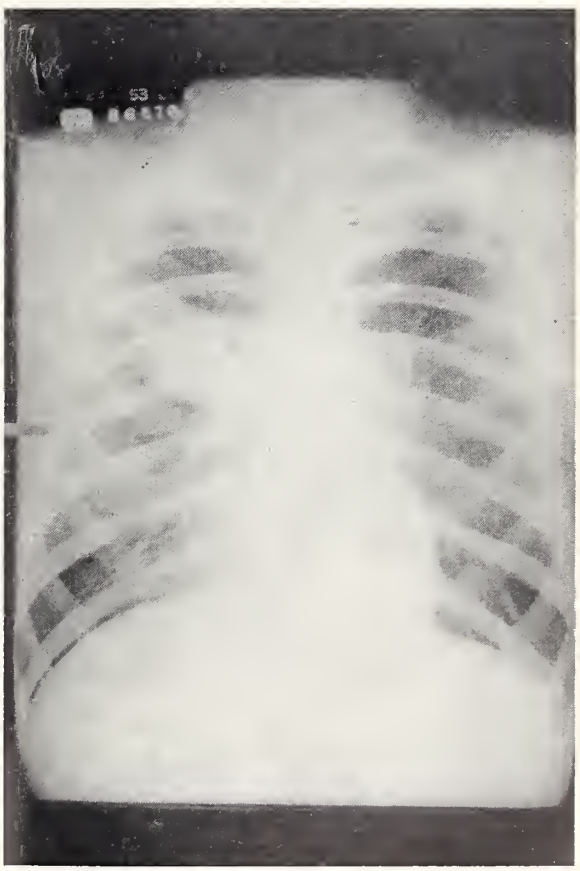


Fig. 4 (D. D.) 32 year old white male showing radio-opacity in right upper chest which proved to be a pulmonary cyst.

CASE REPORTS*

Case No. 1 (S. M.)

This white female was 29 years old when the original chest film made on 2-17-51 showed a cyst at the base of the right lung. She had a history of cough and expectoration each winter since a severe case of pneumonia at age of 15. The original x-ray film showed a very slight amount of fluid in the cyst and no particular reaction around it (Fig. 1). It was decided to treat the patient conservatively at that time. Subsequent films showed a fluid level in the cyst and an increase in the thickness of the wall probably due to pneumonitis, and surgical removal was decided upon (Fig. 2).

Thoracotomy was done on 4-27-53, some two years after the cyst was originally discovered. The cyst was adherent to the posterior chest wall, lying very superficially in the lower lobe and was opened and dissected out, much like a hernial sac. Several small bronchial communications were closed. The patient's convalescence was essentially uneventful and follow-up several months later showed almost complete elimination of cough and expectoration. The post-operative x-ray is shown in Fig. 3.

PATHOLOGICAL REPORT

Specimen consists of a cyst which has been collapsed and in the collapsed state measures 5.5 x 4 x 2.5 cm. The outer surface is brown and in some areas is irregular and roughened while in other portions it is smooth. The cyst lining measures 1 to 2 mm. in thickness with a smooth inner surface. The wall measures 1 mm. in thickness in some areas while in other areas compressed atelectatic lung tissue is present measuring up to 8 mm. in thickness. Histological sections are those of lung in which a cyst is present in the central portion in which the overlying epithelium is absent in most areas but can be identified as being tall columnar respiratory epithelium in others. The wall of the cyst is composed of dense collagenous fibrous tissue in which remnants of bronchi and lung parenchyma are seen. More peripherally there is more compressed atelectatic lung tissue in which dilated bronchi are noted lined with columnar epithelium.

Diagnosis: Cyst of the lung with surrounding compression atelectasis and bronchiectasis. The pathol-

* Two of these three cases were first seen and diagnosed by Dr. G. S. Voorhees, Leavenworth, Kansas.

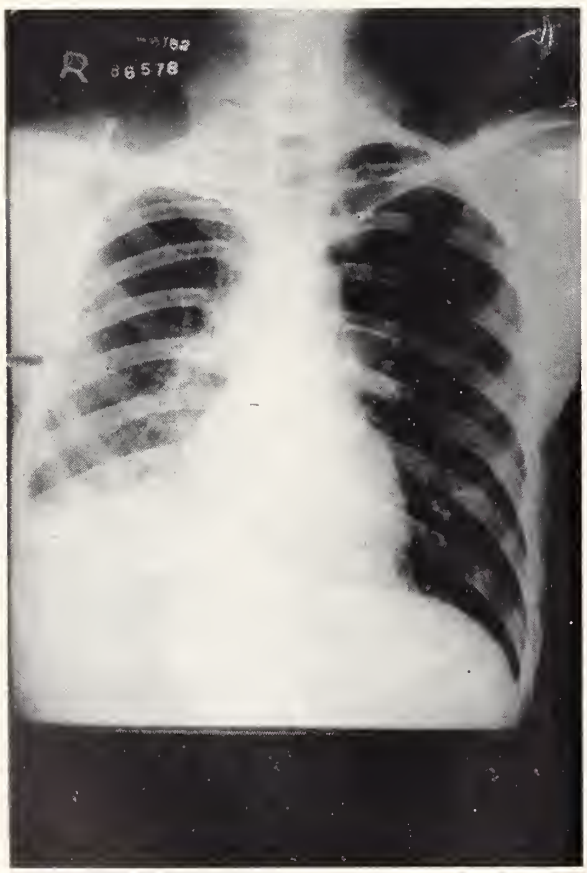


Fig. 5 (D. D.) Same patient after a right upper lobectomy.

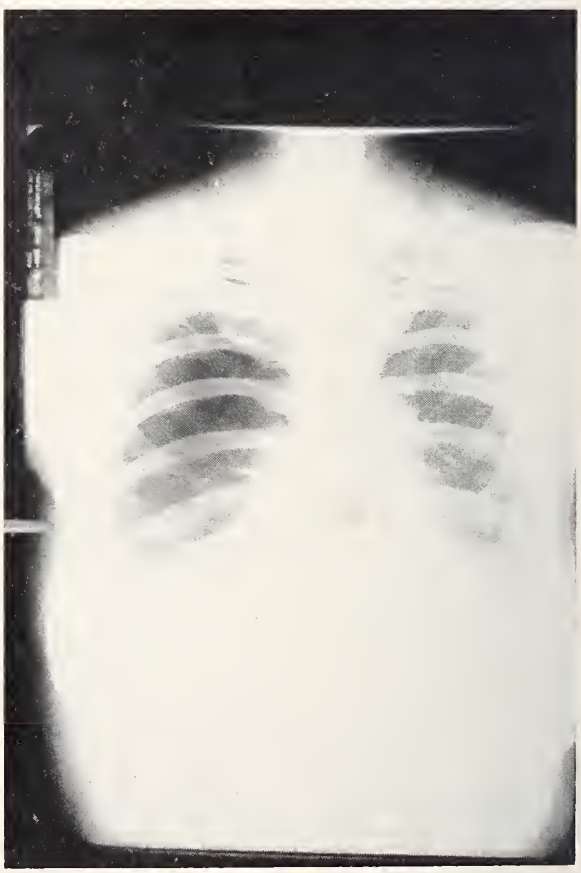


Fig. 6 (B. L.) 42 year old white female showing almost complete absence of right upper lung fields.

ogist makes the comment that "the etiology of this cyst cannot be determined by microscopic section. The histological pattern would fit the diagnosis of a congenital cyst although an acquired cyst cannot be excluded."

Case No. 2 (D. D.)

This 32 year old white male was admitted to the Veterans Administration Hospital, Wadsworth, Kansas, on 9-28-53. One month previously a mass chest survey in his town by the State Public Health Service revealed a lesion in his chest, and hospitalization was advised by his local physician. He had been completely asymptomatic previously, although three years before a routine chest x-ray also apparently revealed a lesion, for the patient was advised at that time to consult his local doctor. The only significant past history was one attack of pneumonia at age 19. Tuberculin, coccidiodin, and histoplasmin skin tests were negative. Bronchoscopy was negative.

The x-rays (Fig. 4) were reported as follows: "The right lung field shows a homogenous lobulated density measuring approximately 8 cm. in its greatest diameter and extending into the lung parenchyma from the right hilar area which appears to lie on the lateral film at approximately the junction of the anterior and middle mediastinum. The possibilities here could be those of a pulmonary aneurysm or an arteriovenous fistula. One must also consider a neurofibroma, dermoid cyst, hamartoma or lymphosarcoma."

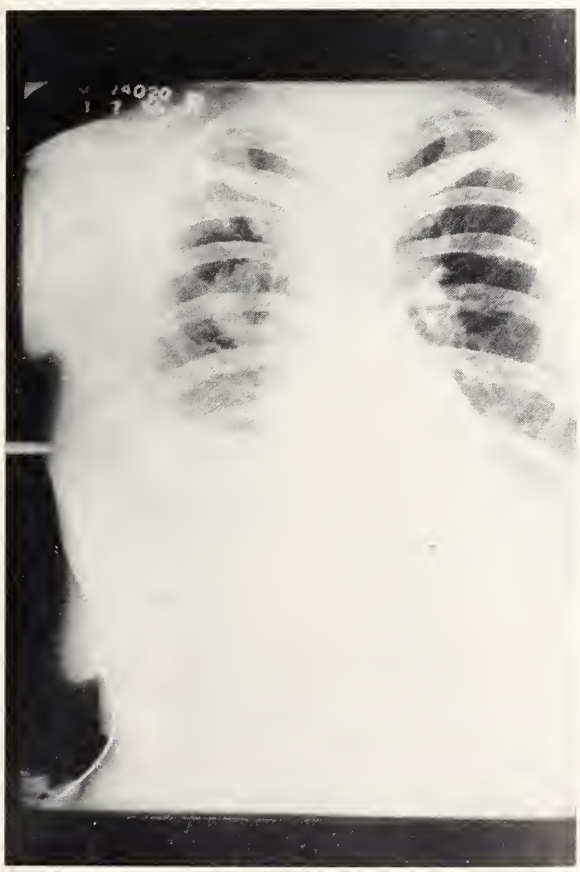


Fig. 7 (B. L.) Same patient postoperatively after extirpation of huge air-filled cyst. The right upper lung field markings are now apparent.

At operation, a cystic structure attached to, but not communicating with the right upper lobe main bronchus, was found and upper lobectomy was performed. The cyst contained viscous and grumous material. Postoperative course was uneventful except for an atelectasis which was relieved by bronchoscopy. The postoperative x-ray is shown in Fig. 5.

PATHOLOGICAL REPORT

The specimen consists of tissue labeled right upper lobe of lung. On palpation one can feel a rather firm mass within the depth of the lobe. On section this is shown to be a multiloculated, cystic cavity which measures approximately 6 cm. in its greatest diameter. The cavity is lined with a thickened, fibrous appearing yellowish gray membrane which is roughened in many areas. Multiple dissections into smaller bronchi in the area and the lobe reveal no definite gross connections between bronchi or bronchioles and the loculated cyst.

Histological sections show a thick walled cystic space that is lined in some areas with intact remnants of respiratory epithelium. Throughout much of the section, however, the epithelial lining is absent, and the surface is covered with pseudo-granulation-like-tissue containing large numbers of lipid laden macrophages. The wall is quite thick, fibrous, and shows infiltration with lipid laden macrophages and mononuclear leukocytes. Smaller bronchioles in the cyst wall show considerable dilatation and are surrounded with cuffs of lymphocytes. On the basis of histology alone, a diagnosis of bronchiectasis was entertained.

Case No. 3 (B. L.)

This 43 year old white female had a large air-filled cyst discovered on a routine chest survey. After the lesion had been found, interrogation of the patient revealed that she had noticed progressive dyspnea on exertion for a period of six months. She was a trained, though not professional, singer and required considerable vital capacity which she noticed to be somewhat reduced during this period. This is understandable on viewing the x-ray film which shows obliteration of the right upper lung fields. (Fig. 6)

Thoracotomy done on 12-9-53 revealed a large air-filled cyst arising from the anterior portion of the upper lobe. It was adherent to the visceral pleura in several areas and its attachment to the upper lobe was almost like a pedicle. It was excised and a few small communicating bronchioles closed. There was

a small emphysematous bleb nearby which was also excised. The patient's convalescence was uneventful. The postoperative x-ray is shown in Fig. 7.

PATHOLOGICAL REPORT

Material consists of a distorted mass of lung tissue measuring 5 cm. in diameter. This ball can be unfolded revealing a flattened sheet of tissue 15 cm. in length with thin walls of fairly uniform thickness. There are some trabeculations on one side of the wall. There is a small amount of lung tissue, and its pleural surface is smooth and glistening and shows some anthracotic pigmentation. Histological sections through the lung show extensive atelectasis and replacement fibrosis. The cyst wall is made of dense fibrous tissue with lymphocytes imbedded in it. The wall is undergoing collagenous degeneration of marked degree, and the lining has largely been denuded.

Diagnosis: Large fibrous walled cyst of the lung with surrounding atelectasis.

COMMENT

These three cases of lung cysts, seen within a period of a year in a general surgical practice, are a suggestion of the increasing frequency with which this disease is being recognized, for it was unknown in American literature less than thirty years ago. These cases are also interesting because they represent three different kinds of cysts.

Case No. 1 is a typical bronchiogenic cyst. Its etiology, however, is conjectural, and whether it should be classified as congenital or acquired would depend upon the authority consulted. The history would, however, offer the possibility of the cyst having an acquired origin. The severe pneumonia at fifteen years of age could have been followed by epithelization of an abscess cavity, since all of the patient's symptoms seem to date from this time. This can, of course, be only speculation for the patient had no x-rays prior to that time.

Case No. 2 seems to definitely fulfill a congenital or developmental origin for it consists of a lesion having bronchial elements that does not communicate with the bronchial tree and is similar to the congenital bronchiogenic cysts of the mediastinum described by Adams and Thornton.[18]

Case No. 3 is a typical alveolar cyst but its etiology is also speculative. The presence of an associated bleb in a singer who undergoes strenuous respiratory activities would seem to point to the possibility that this lesion also arose from a bleb or bulla as has been described by Ackerman and others, and should therefore be classified as acquired.

SUMMARY

The literature on cystic disease of the lung has been briefly reviewed and three case histories described. This is a lesion which is becoming more frequently recognized and in which definitive surgery has been established as the treatment of choice. The classification of the disease and the theories of its etiology, however, still vary considerably with different authorities.

BIBLIOGRAPHY

1. Koontz, A. R.: Congenital Cysts of the Lung, Bull. Johns Hopkins Hosp. 37:340-361, 1925.
2. Eloesser, L.: Congenital Cystic Disease of the Lung, Surg., Gynec. & Obst. 52:747-758, 1931.
3. Wood, H. G.: Congenital Cystic Disease of the Lungs; a Clinical Study, J.A.M.A. 103:815-821, 1934.
4. Schenck, S. G.: Diagnosis of Congenital Cystic Disease of the Lung, Arch. Int. Med. 60:1-21, 1937.
5. Nissen, R.: Development of total pneumonectomy, Am. J. Surg. 78:816-820, Dec., 1949.
6. Graham, E. A. and Singer, J. J.: Successful removal of an entire lung for carcinoma of the bronchus, J.A.M.A. 101:1371-1374, 1933.
7. Rienhoff, W. F.: Pneumonectomy: Preliminary report of operative technic in two successful cases, J.A.M.A. 102:876-877, 1934.
8. Churchill, E. D. and Belsey, B.: Segmental pneumonectomy in bronchiectasis, Ann. Surg. 109:481-499, April 1939.
9. Cooke, F. N. and Blades, B.: Cystic disease of the lungs, J. Thoracic Surg. 23:546-569, 1952.
10. Maier, H. C., and Haight, Cameron: Large Infected Solitary Pulmonary Cysts Simulating Empyema, J. Thoracic Surg. 9:471-494, 1940.
11. Moersch, H. J. and Clagett, O. T.: Pulmonary Cysts, J. Thoracic Surg. 16:179-194, April 1947.
12. Peirce, C. B., and Dirkse, P. R.: Pulmonary Pneumatocele (Localized Alveolar or Lobular Ectasia); Certain Considerations in Cystic Disease of the Lung, Radiology. 28:651-665, 1937.
13. Ackerman, L. V.: Surgical Pathology, C. V. Mosby Co. P. 167, 1953.
14. McAllister, R. V. Maj.: Review of Positive X-ray Findings on Post Photofluoroscopic Survey. Professional Proceedings, Vol. II, Number I:134-138. U. S. Army Hospital, Ft. Leavenworth, Kansas. January 1954.
15. Corpe, R. F., Alexander, J. L., Stergus, I., and Cuadrado, A.: Giant Pulmonary Cyst Simulating Spontaneous Pneumothorax, American Surgeon. 17:753-759, August 1951.
16. Dickson, J. A., Clagett, O. T., and McDonald, J. R.: Cystic disease of the lung and its relationship to bronchiectatic cavities; study of 22 cases, J. Thoracic Surg. 15:196, June 1946.
17. Allbritten, F. F. and Templeton, J. Y.: Treatment of Giant Cysts of the Lung, J. Thoracic Surg. 20:749-760, November 1950.
18. Adams, W. E. and Thornton, T. F. Jr.: Bronchiogenic cysts of mediastinum with report of 3 cases, J. Thoracic Surg. 503-516, August 1943.

The use of a thing is only a part of its significance. To know anything thoroughly, to have the full command of it in all its appliances, we must study in on its own account, independently of any special application. —*Goethe*

Immunity and Resistance in Poliomyelitis

A BRIEF RÉSUMÉ OF THE LITERATURE

Herbert A. Wenner, M.D.

Kansas City, Kansas

A résumé of some old and some new information on poliomyelitis is timely inasmuch as physicians will be called on during the coming months for advice concerning active immunization. Many questions will arise. Some can be, and others cannot be answered. In this brief review I have attempted to summarize some of the opinions on immunity and resistance in poliomyelitis, in a hope that they will be useful in discussions by physicians in the practice of medicine.

HISTORICAL NOTE (1)

Poliomyelitis has undoubtedly occurred in antiquity, and yet periodic outbreaks have been clearly recognized only during the last eighty years. During the last fifty years many have studied the disease. Heine, Medin, and others described the clinical patterns which we recognize at the present time. The essential changes in the central nervous system (CNS) were described by Charcot and by Rissler and by Wickman. Landsteiner and Popper reproduced the disease in monkeys and it was subsequently established that the infectious agent was a filterable virus. Wickman's studies, made during early epidemics, indicated that the disease was infectious and transmissible from person to person. He recognized minor illnesses associated with paralytic poliomyelitis in members of the same household, and considered them to be abortive forms of poliomyelitis. This much had been recognized in 1910.

At this time poliomyelitis entered into a historical era (1910-1930) compounded in error. It was a time when it was considered that poliomyelitis was spread by droplets laden with virus, and the portal of entry was the nasopharynx. It was a time when immune serums were prepared and were found unavailing in the treatment of paralytic poliomyelitis. It was a time when the strict neuronotropism of the poliomyelitis virus was established. Reports appeared in this country and in Sweden that poliomyelitis virus was present in the feces of paralyzed children.[1,2,3]

Many changes occurred during the last twenty years. Old concepts were abandoned for new. Concepts on epidemiology were sharpened. The presence of virus in the gastro-intestinal tract was reconfirmed. Its persistence in the intestine for months after onset of illness, and the potential risk of fecal carriers in populations at risk were recognized. The virus was detected in the oropharynx during the early days of a paralytic attack. Specific antibodies were often found at the onset of paralysis, providing confusion as to the relationship of these antibodies to the disease. Ecologic studies, seeking an extra-human reservoir of virus incriminated the blow fly and certain other species as carriers, if not a means of dissemination of poliomyelitis virus. Antibodies were detected in the serums of cattle and dogs. But the virus was never found in the body tissues of any animal, excluding man. Three immunologically distinct types of poliomyelitis viruses were identified. The virus was detected in the circulating blood—and notions of strict neuronotropism had to be revised. Vaccination tried earlier by Kolmer[4] and Brodie[5] was reconsidered, and brought at the doorstep of clinical field trial.

CHANGES ENCOUNTERED IN THE DISEASE PATTERN

In the Jeremiah Milbank report[1] it is stated—"No circumstance in the history of poliomyelitis is so baffling as its change during the last two decades of the nineteenth century from a sporadic to an epidemic disease." The older literature contains references to localized outbreaks but there is not a single reference to any widespread epidemic. What is the explanation? Was there a change in the virus? Or was there a change in the susceptibility of children since they in particular bore the brunt of the attack?

There is no convincing evidence that poliomyelitis suddenly acquired virulence permitting it to provoke clinical illness. Nothing exists to indicate that the infection was caused by a virus introduced into a highly susceptible population. Indeed, the fact that children and not adults were attacked most often, suggests that adults had acquired immunity as a result of previous experience with poliomyelitis virus. It is possible that a mutant strain possessing enhanced virulence emerged and suddenly became dominant in a population of viruses heretofore almost entirely harmless in human beings, but it is not likely.

What changes occurred among people? It was a period of changes; some of these are still in evolution. There were *social changes,* among them the trend toward smaller family units. There was a migration of people from the town and country to the city. There were changes in *concepts of hygiene.* Pasteur's microbes led to the Listerian era and thence

From the Department of Pediatrics and the Hixon Memorial Laboratory, The University of Kansas School of Medicine, Kansas City, Kansas.

From a paper presented before the Erie County Academy of General Practice, Buffalo, New York, April 9, 1954.

to community health. In the cities, slops and dung were disposed of by underground sewers. It was not perfect. Privies and other local deposits of ordure remained, as they do today in some of our urban and suburban areas. There were also changes in family hygiene. Houses were screened against flies. Efforts were put forth to stamp out infestation of homes and industrial places of rats and insects. The individual became aware of the danger of germs and personal hygiene probably reached as high a level during the last half of the nineteenth century as it had for the Greeks and Romans at the height of their respective civilizations.

The first large epidemics appeared in Sweden, and shortly thereafter in North Central Europe and in the United States. Widespread outbreaks soon appeared in north and south temperate climates. By and large, peoples living in the tropical and semitropical countries were spared outbreaks of epidemic proportions, but sporadic cases appeared more or less constantly. Soldiers from temperate climates on entering the tropical areas were not spared from poliomyelitis although very few cases were seen among the native population. It is assumed that the rebirth of community hygiene, chiefly in the form of modern water and sewage disposal plants, has commanded a fee best calculated in loss of immunity to poliomyelitis because less and less virus was available for immunization during infancy, and for periodic reinforcement during later life. A contrasting situation exists in certain tropical and semitropical areas where it is difficult for an informed person, and practically impossible for an uninformed person, to escape fecal contamination. Hence, rapidly acquired latent immunization, and constant reinforcement of the antibody mechanism, protect the people in these areas from frank clinical forms of poliomyelitis.

Two other changes will be noted, namely, (a) in age groups attacked by poliomyelitis, and (b) in rural versus urban attack rates. At the beginning, infants and young children were almost always the victims of paralysis. Over the past thirty-five years there has been a gradual inclusion of more teenagers and young adults. In the early years of the twentieth century there were more older children and young adults attacked in rural areas. At the present time the age incidence in town and country are not different. This interesting shift in age distribution is not well understood. It is of serious portent because young adults, in comparison with children, have a more severe illness, are apt to have bulbar involvement, recuperate less rapidly, and the death rate is two or three times higher.

The clinical attack rates in rural and suburban populations have exceeded those observed for the more densely populated sections of cities. Forty years ago these changes were striking enough that Lavender, Freeman, and Frost[1] considered "the proportionately greater rural prevalence has been so constant as to be unmistakably due, not to mere chance, but to some fundamental law." An explanation given to these findings has been that latent immunization proceeds faster in urban that it does in rural areas, providing in the latter accumulations of susceptibles, and hence proportionately more at risk of the paralytic event. There are other factors related to means of virus spread including the possibility of extra-human reservoirs of poliomyelitis virus in the animal kingdom. During recent years in some sections of the United States the urban and rural attack rates are no longer very different.

In summing up, it is clear that many changes have occurred in the disease pattern of poliomyelitis since 1880. Some evidence suggests that these changes are related to host-susceptibility, chiefly because of rapidly changing concepts in socio-economic conditions and that these have had an impact on family and community life. It would appear probable that with improved sanitation, and the consequent removal of the largest source of virus in sewage, that latent immunization proceeds at a slower rate, and that there is a discontinuous distribution in age-specific attack rates with a continuing accumulation of susceptibles available for infection with the virus. Others have considered that resistance may be related to either (a) genetic factors of susceptibility, or (b) seasonal alterations in the intracellular milieu which facilitate invasion and disease.

THE PATTERN OF POLIOMYELITIS IN THE INDIVIDUAL

The diagnosis of paralytic poliomyelitis is not difficult, and seldom challenges the acumen of the clinician. It is quite difficult, if not impossible, to make the diagnosis prior to the onset of signs related to the central nervous system. The pre-paralytic phase of illness has been described, but is not widely appreciated. It consists of fever, malaise, sore throat, diarrhea, and abdominal pain, lasting two to four days. There may then be a comparatively symptom-free interval of several days before paralysis intervenes. It is well recognized that many individuals experience mild prodromata, and that the intervening paralysis appears apparently without antecedent signs or symptoms. Further, many individuals who have the pre-paralytic type of illness never have subjective evidence of paralysis, and it is now ascertained that these illnesses occurring in an epidemic area, particularly in relationship to associate cases, are abortive forms of poliomyelitis. The ratio of the paralytic and non-paralytic forms have been estimated as being from 1:1000 to 1:20.

THE PATTERN OF POLIOMYELITIS IN THE HOUSEHOLD

The existence of minor illnesses which undoubtedly represented infection with poliomyelitis virus was suspected since the early studies of Wickman, Caverly, and others.[1] Definite proof that these illnesses

were poliomyelitis was obtained by Trask, Paul, and their co-workers,[6] and in recent years by Francis, and by Wenner and their colleagues.[7, 8] In certain families, particularly those with many young children, it is the rule rather than the exception to obtain a history of non-paralytic illness in several members associated with a paralyzed sibling. Proof that these illnesses are caused by poliomyelitis virus has been obtained by the direct isolation of virus from the stool, and by the emergence of type-specific antibodies in the blood stream of these individuals.

In the past, as now, it has not always been easy to determine whether one of the family members introduced the virus into his household, or whether the family group was infected from some common source. Our family studies have led us to the following conclusions. First, the onset of illness among familial associates in relation to the index case occurred three or four days before or after onset in the paralyzed patient. Considering the mean incubation period to be about twelve days,[9, 10] this means that members of this family unit were infected at a common source. It is our opinion that a food product contaminated by poliomyelitis virus is the logical source. Admittedly proof for this assumption, excepting possibly for milk-borne outbreaks, is hard to obtain. A study by Zintek[11] in which it was shown that virus appeared simultaneously in the feces of all members of a household associated with a paralyzed child add support to the assumption that parasitism of the alimentary tract occurs presumably from virus acquired at a common source.

Multiple household infections are not common to all families. As children become older and migrate into extra-familial groupings it is logical that isolated cases can occur. We have studied two such families in which only one member, each in his early teens was the only member in the family demonstrated to carry virus. These individuals in daily contact with their family associates failed to infect them, indicating to us that infection with poliomyelitis virus is not readily acquired by droplet infection. Unfortunately, these studies were done several years ago when simple, inexpensive tools for serum antibody levels were not available. The antibody levels before, during, and after exposure are essential in support of our assumptions. We are engaged in obtaining this information at the present time.

There are instances when secondary cases may appear within affected households. A sibling playmate of a neighbor's child acquired his infection from the latter. The sibling in the prodromal period infected his brother. It is our opinion that infection may occur as a result of oral ingestion of virus presumably originating in feces containing poliomyelitis virus. Consider the readiness with which pinworm infestation occurs in children and adults in a household! It has been, and continues to be fairly easy for infected adults to disseminate enteric disorders. How simple then is it for a child to pass feces containing poliomyelitis virus from his fingers to the oral cavity of others who play or care for him.

THE PATTERN OF POLIOMYELITIS IN THE COMMUNITY

A low paralytic attack rate occurs in poliomyelitis. In severe outbreaks it is uncommon to have over-all urban rates exceeding 4/1000 population; it is not uncommon to meet rates higher than this in rural communities. The low rates are somewhat misleading in that they fail to be concerned with rates of infection, a subject on which more information is needed. It would appear that low paralytic rates may be attributed to the following: (a) a high index of infection with a low index of disease, and a resulting high index of immunity in the general population, (b) a high index of infection in stratified segments of the population with corresponding segmental immunity which is not nearly so comprehensive as measles or influenza in the total aggregate of people attacked, or (c) a high index of infection in the population at risk, the paralytic event occurring as a result of an anatomical accident.

The ratio of subclinical to clinical infections is considered to be a function of age.[12] Serologic surveys indicate that the rate of antibody acquisition is not constant since the distribution of antibodies in 5-year age groups varies considerably in different geographic areas. In some urban areas of the United States at age 10 (years) about 50 per cent, and at age 20 about 75 per cent of individuals acquire antibodies against one or more of the known types of poliomyelitis viruses. Were these data to be translated to the country as a whole, acquisition of virus by the population at risk would be fairly rapid and comprehensive.

If one assumes that the ratio of inapparent and apparent infection is constant, one cannot be other than perplexed at the irregularities in the geographic location of cases occurring in urban areas. The studies made by Francis et al,[7] and by Wenner et al[8] point out that there is a tendency to have aggregation of cases around infected households. Francis has said, "The major concentration of poliomyelitis virus is in the families, including adults, and in intimate groups in which the disease appears. . . ." The available evidence lends support to the notion that the velocity of spread of poliomyelitis virus is slow, and that over the summer and autumn months irregular foci of cases (and those inapparently infected) appear and gradually converge on each other, providing what would appear to be uniform dissemination of virus when actually a small and limited fraction of the population has been involved. The slow radial movement of poliomyelitis has been noted. In Casey's study the rate was 0.1 mile per day; in Paul's study it was 0.5 mile per day.

These observations suggest that only a part of the population, chiefly at risk, namely children under 10 years of age, have been infected with poliomyelitis virus, and that a large and relatively unknown segment remains unaffected during any given season and awaiting subsequent attack. Our studies suggest that the velocity of spread is slower than measles, for example, because the virus requires a medium of transfer similar to that of enteric diseases, and that the infection rates are lower than measles leaving behind many more susceptibles, some of whom will become immune by virtue of living in an environment periodically and sporadically seeded with virus.

THE PATTERN OF IMMUNITY IN THE INDIVIDUAL

Recurrent attacks of paralytic poliomyelitis occur; the chance of reoccurrence is infrequent. It was thought that an attack of poliomyelitis conveyed an appreciable immunity to a new infection. The serologic response, often used to indicate immunity, was rather disturbing because antibodies were present in the patient's serum virtually at the time that paralysis made its appearance. Further, no appreciable increase occurred during convalescence. Hence it was difficult to know whether poliomyelitis antibodies were present because of the present infection or were the result of a previous experience with the virus or a related antigen. In a small proportion of the cases there was a relatively slow rise of neutralizing antibodies over a period of several months.

At the present time some of these difficulties have been solved. It is recognized that illness caused by poliomyelitis virus occurs about a week prior to onset of central nervous system involvement. Moreover, viremia occurs during this period in a substantial number, if not all, of the people who experience primary infections. Hence neutralizing antibodies are in the blood stream at the time when injury to the central nervous system is manifest. The recognition that there are three distinct immunologic types of poliomyelitis viruses, and that they appear to be mutually exclusive in the specificity of the antibody response, offers an explanation why antibodies aren't found in some patients—their serums were tested usually with a heterotypic strain of virus. Only in recent years have serums obtained from patients been tested with the patient's virus.

The acquisition of antibodies against the three known types increases with age. At age 5, for example, 20 to 40 per cent of children in various areas of the United States have antibodies against one type, while at age 18, about 40 per cent have antibodies to all three types. Many of these individuals have never been ill with paralytic poliomyelitis. Those who have had paralytic poliomyelitis have had but a single attack. What this means is that infection with the other heterotypic viruses was unrecognized, which is probable, or that there is antigenic overlap among certain strains, which appears at the present time somewhat improbable.

In summary, paralytic poliomyelitis conveys homotypic immunity. Such immunity is evidenced by resistance to the disease (but not necessarily to infection with another type) and by the appearance of homotypic humoral antibodies which persist for years. A large number of abortive infections occur with poliomyelitis virus and obviously convey an immunity durable enough to prevent the paralytic form of the disease.

THE PATTERN OF IMMUNITY IN THE COMMUNITY

In regard to community epidemiology we remarked on the slow velocity of infection, and the focal accumulation of cases in households and in intimate groups. Unfortunately, at this time there is limited evidence from serologic studies in support of, or in opposition to, these observations. A large number of serologic studies have been made in several communities. Before the tissue culture methods became available the type 2 (Lansing) virus was used because tests could be made on a statistically sound basis using mice. A large amount of information exists in regard to the distribution of type 2 neutralizing antibodies. In Baltimore, the emergence of Lansing antibodies was observed to occur during the summer months. By the time an individual is 10 years of age, and if he resides in the Eastern Health District of Baltimore[13] he has acquired type 2 antibodies. In Cario, Egypt antibodies are found in 70 per cent of three year olds, and in all by the time children are four years of age.[14] Among Alaskan Eskimos antibodies were absent in individuals less than 20 years of age, and were present in practically all individuals of 20 years or older.

Samplings in regard to the presence or absence of antibody have been made in various geographic areas and in various age groups and with regard to socioeconomic factors. Although these data seem to indicate universality of infection at a given age in a population, they have failed to provide an index of velocity of infection. The only pertinent report on the aspect is by Melnick and Ledinko, whose studies in Winston-Salem began before and continued beyond an epidemic period. By following the development of neutralizing antibodies in 200 children, they showed the emergence of antibodies against type 1 and type 2 viruses, and none against type 3 poliomyelitis virus. On the basis of these data, they were able to determine the ratio of subclinical infections to paralytic cases. These varied somewhat with age, ranging from 175:1 for infants less than a year of age, to 95:1 for those 10 to 14 years of age. It would appear from this study that in any given epidemic year about one-fourth of the population under 15 years of age becomes immunized. Since latent immunization occurs between epidemics some

of the remainder are being continuously added to the immune group. On this basis poliomyelitis has a rather sweeping inclusion of susceptibles in epidemic years, particularly in regard to the specific type of virus responsible for the outbreak. At this time it is necessary to recall that during a number of outbreaks several types may be isolated in a given community.

PATTERNS DESIGNED FOR THE CONTROL OF POLIOMYELITIS

The available data indicate that in a population many are at risk of infection, but few are at risk of paralysis. The risk obviously diminishes with age, although this is not a constant, since older age groups are attacked somewhat more frequently in the last thirty years. The occurrence of paralysis in poliomyelitis is an intriguing problem in pathogenesis, and at the present time all of the reasons for its occurrence are not known.

Three approaches have been considered in immunization against poliomyelitis. The approaches are based, in part, on facts discussed earlier in this paper. They include (a) a "killed" virus vaccine, (b) active virus vaccine, with or without administration of antibody, and (c) an avirulent virus vaccine.

The formalin-treated triple vaccine is an example of the first type, and is the kind of vaccine which is currently being made ready for the field vaccination trials. The use of killed virus vaccines has disadvantages in respect to the development of immunity and the endurability of the immunity. The premise on which vaccination with formalinized virus is based, is that (a) the humoral antibody level need not be high (i.e. about 1:64) in order to block the passage of poliomyelitis into the central nervous system, and (b) the augmentation of the antibody response will occur periodically as a result of re-infection with all three types of poliomyelitis viruses. If the premises are correct, vaccination with formalin-inactivated viruses may turn the tide away, not from infection with poliomyelitis viruses, but from the risk of the paralytic event.

The formalinized vaccine has been studied rather extensively in Allegheny County, Pennsylvania. The antibody response using the polyvalent vaccine in individuals who do not have antibody is low in magnitude (i.e., about 1:16 to 1:64). The response in individuals who do have antibody is swift and the level of antibody reaches high levels (i.e., about 1:1000 or better). The important issue in the field trial is whether antibodies acquired by non-immune individuals is of sufficient level to prevent the 1:100 to 1:1000 risk of paralysis.

An issue of importance is whether vaccination with formalin-treated virus will create an enduring immunity. Will vaccination with "killed" virus be an annual event among children? The answer isn't known. If re-vaccination becomes necessary what are the risks involved in the repetitious use of killed virus grown in monkey epithelial cells? The answer to this question isn't known either, although available information indicates that the vaccine has no nephrotoxic effect as currently used. Several of us have raised the query: What effect will the administration of monkey kidney cell debris have on the development of Rh antibodies? It is believed that monkey tissue and red cell antigens are different and that the risk of sensitization in Rh negative girls is improbable. The belief has not yet been examined in the test situation, and the answer is not available at this time.

Immunization with active virus together with gamma globulin has a number of disadvantages. First, passive antibody ties up the virus and active immunity is unlikely to follow parenteral injection of virus. A much more serious objection is that antibody-virus mixtures are not always inactivated and active virus may produce infection in the presence of antibody. From the point of view of practicability of such type of vaccination, it is not safe.

Active immunization may follow oral administration of virus in human beings. Koprowski[15] and his associates have fed a type 2 strain of rodent-adapted virus to children. Following oral administration of virus there was a significant rise in type 2 antibodies. None of the children suffered any ill effects.

A number of laboratories are searching for avirulent strains of poliomyelitis viruses. There is some evidence to indicate that they occur. The adaptation of strains to hamsters and then to chick embryos[16] has resulted in a variant which has lost much but not all of its original virulence. The passage of certain strains, using special techniques in tissue culture, has modified strains so that their behavior does not resemble the effect of the parent strain in experimental animals. None of these strains is ready for test in human beings. There is sufficient promise in the work already done to urge further studies in the effort to obtain modified poliomyelitis viruses, avirulent for monkeys, but on oral or parenteral administration capable of providing immunity to poliomyelitis in human beings.

BIBLIOGRAPHY

(A detailed bibliography on this résumé is not given here for purposes of brevity.)

1. Poliomyelitis. A survey made possible by a grant from the International Committee for the study of infantile paralysis organized by Jeremiah Milbank. The Williams and Wilkins Co., Baltimore, 1932. Early refcrences arc listed in this monograph.
2. Kling, C., Patterson, A., and Wernstedt, W.: Tbe presence of tbe microbe of infantile paralysis in human beings. Communications Inst. méd. État, Stockbolm 3:5, 1912.
3. Sawyer, W. A.: An epidemiological study of poliomyelitis. Am. J. Trop. Dis. 3:164, 1915-16.
4. Kolmer, J. A., Klugh, G., Jr., and Rule, A. M.: A successful method for vaccination against acute anterior poliomyelitis; further report. J.A.M.A. 104:456, 1935.
5. Brodie, M., and Park, W. H.: Active immunization against poliomyelitis. J.A.M.A. 105:1089, 1935.
6. Paul, J. R., Salinger, R., and Trask, J. D.: Studies on the epidemiology of poliomyelitis. II. The incidence of abortive type of poliomyelitis. Am. J. Hyg. 17:601, 1933.
7. Pearson, H. E., Brown, G. C., Rendtorff, R. C., Ridenour,

G. M., and Francis, T., Jr.: Studies on the distribution of poliomyelitis virus. III. In an urban area during an epidemic. Am. J. Hyg. 41:188, 1945, also, Trans. Assoc. Am. Physicians 65:176, 1952.

8. Wenner, H. A., and Tanner, W. A.: Poliomyelitis in families attacked by the disease. I. Distribution of virus in stool and oropharynx of members of households. Am. J. Med. Sc. 216:258, 1948.

9. Casey, A. E.: The incubation period in epidemic poliomyelitis. J.A.M.A. 120:805, 1942.

10. Sartwell, P. E.: The incubation period of poliomyelitis. Am. J. Pub. Health 42:1403, 1952.

11. Zintek, A. R.: The rapid infection of a family after introduction of poliomyelitis virus. Am. J. Hyg. 46:248, 1947.

12. Melnick, J. L., and Ledinko, N.: Development of neutralizing antibodies against the three types of poliomyelitis virus during an epidemic period. The ratio of inapparent infection to clinical poliomyelitis. Am. J. Hyg. 58:207, 1953.

13. Turner, T. B., Young, L. E., and Maxwell, E. S.: The mouse-adapted Lansing strain of poliomyelitis virus. IV. Neutralizing antibodies in the serum of healthy children. Am. J. Hyg. 42:119, 1945.

14. Paul, J. R.: Geography and antibodies. Trans. Assoc. Am. Physicians 65:184, 1952.

15. Koprowski, H., Jervis, J. A., Norton, T. W., and Nelson, D. J.: Further studies on oral administration of living poliomyelitis virus to human subjects. Proc. Soc. Exp. Biol. and Med. 82:277, 1953.

16. Cabasso, V. J., Stebbins, M. R., Dutcher, R. M., Moyer, A. W., and Cox, H. R.: Poliomyelitis. III. Propagation of MEF-1 strain of poliomyelitis virus in developing chick embryo by allantoic cavity inoculation. Proc. Soc. Exp. Biol. and Med. 81:525, 1952.

(Some other references not cited specifically)

Bodian, D.: A reconsideration of the pathogenesis of poliomyelitis. Am. J. Hyg. 55:414, 1952.

Hammon, Wm. McD.: Immunity in poliomyelitis. Bact. Rev. 13:135, 1949.

Sabin, A. B.: Present status and failure possibilities of a vaccine for the control of poliomyelitis. Am. J. Dis. Child. 86:301, 1953.

Salk, J. E.: Principles of immunization as applied to poliomyelitis and influenza. Am. J. Pub. Health 43:1384, 1953.

Rupture of the Colon Secondary to Muscular Strain

REPORT OF TWO CASES

W. G. Cauble, M.D.*

Wichita, Kansas

Rupture of the colon secondary to muscular strain is rare and results in a severe abdominal catastrophe. It is thought that it occurs usually as a result of some underlying pathology. Rupture of the bowel under an intact abdominal wall has long been a subject of interest to the surgeon, mainly because of the acute severity of the condition.

Wilensky and Kaufman[8] reviewed the literature up to 1936 and found forty-two cases of ruptured intestine due to muscular effort reported at that time. They reported one additional case. In the majority of these cases the rupture occurred secondary to a hernia and involved in most cases the small bowel. It is interesting to note that some of the early writers thought that increased intraperitoneal pressure could not, in itself, result in intestinal rupture. It has been shown that adhesions and agglutinations of the intestines predispose to rupture after severe straining by inhibiting the motility of the bowel. Berman *et al*[1] in further discussing bowel perforation with hernia as a secondary cause stress the importance of this condition in industrial medicine. In one of their two cases reported, compensation was allowed while the second one was in arbitration at the time the article was written. Several cases of rupture of the colon due to compressed air have been reported by writers.[2, 3, 4, 5, 6] This type of rupture is brought about by the introduction of compressed air into the rectum—usually during some "horse play" or prank. Prior to the use of antibiotics the mortality rate of these cases was quite high. We are all acquainted with the fact that perforation of the colon can be brought about while the patient is getting a barium enema or by the introduction of the colon tube. Rupture of a diverticulum of the colon is not uncommon, as is perforation in ulcerative colitis.

Weinstein and Roberts[7] in 1952 reported one case of spontaneous perforation of the colon without a pathological condition. Up to that time only one other similar case had been reported according to their article. Their patient perforated while having a bowel movement. Qvigstad[9] reported a case of perforation of the transverse colon in a four day old infant. An autopsy was done and microscopy revealed a defect in the muscular layer at one side of the perforation. He thought that the pathogenesis possibly could have been a congenital anomaly.

The following two cases are being reported mainly because of the rarity of the condition. The first case, with the perineal stoma following a Lynch operation for carcinoma of the rectum would questionably have some underlying pathology, while the second case could definitely be secondary to some long standing pathology.

CASE NUMBER ONE

Mr. C. L. P., a 58-year-old, white male was admitted to St. Joseph Hospital, November 13, 1951, with the chief complaint of severe pain in the lower abdomen. At 5:00 A. M. that morning the patient had gotten out of bed and went to the bathroom to have his usual morning bowel movement and while straining at the stool he developed severe acute pain in the lower part of the abdomen and became unconscious. He gradually regained partial consciousness and was brought into the hospital, traveling a distance of approximately 75 miles.

* From the surgical service of Wichita-St. Joseph Hospital, Wichita, Kansas. Submitted under the auspices of the Wichita Medical Research Foundation.

The past history of the patient revealed that on October 10, 1950, the patient was operated on by Doctor Claude Tucker, of this city, for carcinoma of the rectum. A Lynch operation was done at that time. The pathological diagnosis was Grade II adenocarcinoma of the rectum with metastasis to regional lymph nodes.

Upon admission the patient was semi-conscious, nauseated and vomiting. He appeared to be in shock. His pulse was somewhat weak and was 100 per minute. His temperature was 101.8°. Examination of the abdomen revealed moderate distention with generalized tenderness. There was considerable muscle guard over the whole abdomen with marked rebound tenderness. The perineal stoma was examined and nothing unusual was found. The admission diagnosis was probable acute intestinal obstruction.

Soon after admission a flat plate of the abdomen was done, but this revealed no evidence of any intestinal obstruction. The admission blood count revealed 4,420,000 red blood cells, 13 grams of hemoglobin, 9,850 white blood cells, with 70 segmenters, 1 stab, 2 eosinophils and 27 lymphocytes. The urinalysis was essentially normal. He was given intravenous fluids and antibiotics, with other supportive therapy, and was later taken to the operating room. At operation a right rectus incision was made and the abdomen opened. On examination of the abdomen no evidence of any recurrent carcinoma was found. The patient was very obese, so considerable adipose tissue was encountered in the abdominal wall and in the abdomen. On opening the abdomen a large amount of fecal material was found in the abdominal cavity, which appeared to be coming from the left lower abdominal gutter. A perforation, approximately one-half inch in length, was found posterior to the bladder, in the hollow of the sacrum. Due to the adipose tissue and the position of the lesion, it was impossible to see the area, but the perforation could be felt. In view of this, a transverse colostomy was carried out and a stab wound was made in the left rectus area, through which rubber drains were brought out, draining the pelvis, and the left gutter. Due to the shortness of the colon it was necessary to bring the colostomy out of the upper portion of the incision. The colostomy was a simple loop colostomy. A mushroom catheter was placed in the proximal portion of the colon. Another drain to the right side of the pelvis was brought out the lower portion of the incision. After routine closure the patient was placed in a lithotomy position and a rubber tube drain was placed into the hollow of the sacrum through an incision made posterior to the perineal stoma. He left the operating room in fair condition and was given transfusions.

This patient was given large doses of antibiotics, mainly penicillin, and all drainage tubes were removed in due time. He progressed nicely until the 19th of November, when he had a severe coughing spell and eviscerated. He was then taken back to the operating room where a secondary closure of the eviscerated wound was carried out. Following that procedure he did well and on November 28, 1951, he was returned to surgery and the colon was divided completely, leaving a functioning and nonfunctioning colostomy. The two stomas were approximately three inches apart. Following that he did well and was dismissed from the hospital December 2, 1951. Since dismissal from the hospital the patient has been in good health, has been doing his work, which has consisted of routine farm work and driving a tractor. He has not been seen, but in a recent written communication he stated that he has felt well and is well satisfied with the abdominal colostomy.

CASE NUMBER TWO

S. S., age 42 years, white male, was admitted to St. Joseph Hospital on January 29, 1954. The afternoon and evening prior to admission he had been working hard and had been lifting some Butane tanks which weighed approximately 150 pounds. He had worked until 11:00 P. M. that night and had gone home and gone to bed. At approximately 2:00 A. M. he was suddenly awakened with severe acute lower abdominal pain. He vomited several times and his general condition became worse. He finally called his family doctor and then was admitted to the hospital that afternoon with severe generalized abdominal pain. His past history revealed that he had had a ruptured appendix at the age of nine, at which time the appendix was not removed, but was drained. He was very ill at the time and the drain was left in for several days. He had had no serious difficulty and no surgery other than that at the age of nine.

On examination this patient was acutely ill. The abdomen revealed moderate distention and generalized tenderness, which was worse in the right side. There was considerable muscle guard and rebound tenderness over the whole abdomen. Rectal examination revealed nothing unusual. The admission diagnosis was intestinal obstruction with peritonitis. A flat plate of the abdomen was taken in the supine and upright positions and a diagnosis of probable mechanical intestinal obstruction, involving both jejunal and ileal segments was made. On admission the blood count revealed 5,200,000 red blood cells, 14.5 grams of hemoglobin, 15,850 white blood cells with 77 segs, 8 stabs, 15 lymphs. The urine was essentially normal except for 8 to 10 pus cells per high power field. He was given supportive treatment including intravenous fluids and 500 cc. of whole blood. He was started on antibiotics, mainly penicillin and dihydrostreptomycin.

He was taken to the operating room where the abdomen was opened through a right rectus incision

and upon opening the abdomen considerable free pus was found and aspirated and generalized peritonitis was evident. On examination of the abdominal viscera the pathology was found in the region of the cecum. No appendix was present, but there was apparently a thinned area in the wall of the cecum where the appendix had been and through this thinned out area was a one-half inch perforation. This was closed with catgut sutures and a stab wound was made to the right of the incision and two rubber drains were brought out through that wound. These drained the region of the cecum and the right abdominal gutter. Routine closure was carried out and the patient returned to his room in fair condition. Large doses of penicillin and dihydrostreptomycin were given the patient. A Levine tube was left in the stomach for four days and the drains were removed after five days. His convalescence was smooth and uneventful. He was dismissed on February 12, 1954 and has been seen at intervals in the office. He has done well and when last seen, a month ago, he had no complaints.

DISCUSSION

Rupture of the colon secondary to muscular strain is a rare condition and should be kept in mind when a patient is seen with severe peritonitis, which develops following acute severe abdominal pain. The seriousness of the condition cannot be over-emphasized and the value of large doses of penicillin and dihydrostreptomycin cannot be underestimated.

SUMMARY

1. Review of literature has been presented.

2. One case of ruptured colon following severe straining at the stool has been presented. This patient had had a Lynch operation for carcinoma of the rectum three years previously. At the last operation no evidence of any recurrence of the carcinoma was found.

3. Another case has been presented in which perforation of the cecum followed heavy lifting and straining. The perforation was apparently in the location of the base of the appendix, which had sloughed away when the patient was a child.

4. The severity of the condition has been stressed.

BIBLIOGRAPHY

1. Berman, Harry; Mainella, Frank, and Rosner, Henry: Rupture of the intestines in cases of hernia due to severe muscular strain, Internat. Col. Surgeons 18:122-125 (July) 1952.

2. Brown, Roswell K., and Dwinelle, J. H.: Rupture of the colon by compressed air, Ann. Surg. Vol. 115, No. 1:13-20 (January) 1942.

3. Ide, Arthur W.: Pneumatic rupture of the bowel, Minn. Med. 19:265-268 (April) 1936.

4. Lapp, Harry C.: Pneumatic rupture of the colon, J. Missouri M.A. 42:776 779 (December) 1945.

5. Waugh, Rickey L. and Leonard, Field C.: Rupture of the colon due to compressed air with particular reference to the character of the strain, Mil. Surgeon 108:294 301 (April) 1951.

6. White, R. J.: Injury to the colon from air hose inflation, Texas State J. Med. 41:406 407 (December) 1945.

7. Weinstein, Mondel and Roberts, Martin: Spontaneous perforation of colon in absence of pathological condition, J.A.M.A. Vol. 149:1016-1017 (July 12) 1952.

8. Wilensky, Abraham O., and Kaufman, Paul A.: Subparietal rupture of the intestine due to muscular effort, Ann. Surg. 106 (September) 1937.

9. Qvigstad, Ivan: Spontaneous perforation of the colon in the newborn, Nordisk Medicin No. 12 Vol. 43:504-505 (March 24) 1950.

An Error in Blood Sugar Determinations

Mark Dodge, M.D., Marion Mathy, Arthur W. Robinson, M.D., and Arnold V. Arms, M.D.

Kansas City, Missouri

The diagnosis of diabetes mellitus applied to the individual patient is so important in the latter's economy as regards his way of life and the practical matters of life insurance, etc., that it behooves us to use the most accurate diagnostic methods available for this designation.

We have been impressed by the work of Mosenthal[1] in a review describing the discrepancy between true blood sugar and total reducing substances in the blood. The popularly used Folin-Wu[2,3] method for blood sugar determines the latter whereas the Somogyi[4] technique is supposed to reveal the former. Obviously, if a method is available for the doctor's office and the average hospital laboratory to measure only true blood sugar, it is to be preferred over the Folin-Wu procedure. Therefore, we have set out to determine whether the Somogyi method is technically feasible, accurately reproducible (including recoverability of added glucose), whether after removal of blood glucose by yeast fermentation the Somogyi method reads zero, and whether by the fermentation technique the difference between the total reducing substances and the true blood sugar can be predicted by the Somogyi method.

Having studied these factors, we have determined a number of fasting and non-fasting blood sugars in diabetics using both methods, and for comparison have repeated the determinations on several of the same patients to check for individual variations in non-glucose reducing substance in a particular diabetic over a period of time.

In addition we have documented numerous glucose tolerance tests using both methods dually.

MATERIALS AND METHODS

The Somogyi modification described by Nelson[6] uses a solution of $Ba(OH)_2$ which is unstable, so we chose the method originally suggested by Somogyi:[4]

A. Blood Filtrate
1 cc. oxalated blood dissolved in 7 cc. of H_2O
Add 1 cc. Sol I and mix
With continual shaking add 1 cc. Sol II
Filter

Sol I is 10% $ZnSO_4 \cdot 7H_2O$
Sol II is 0.5 N NaOH
} These must be titrated against each other so that 10 cc. Sol I added to 50 cc. of H_2O will turn phenolphthalein pink with 10.8 cc.—11.2 cc. of Sol. II.

B. 2 cc. of this blood filtrate in a Folin-Wu sugar tube
2 cc. of alkaline copper tartrate solution
Mix with lateral shaking
Boil 8 minutes in boiling water bath
Cool to room temperature
Add 2 cc. phospho-molybdic acid sol and mix
Place in boiling water for 5 minutes
Dilute to 25 cc. with distilled water and mix
Read in Leitz colorimeter set at 520.

It is to be noted that part B is the same as part B in the Folin-Wu method. The strategic reagent is the neutral zinc, which causes precipitation of the non-glucose reducing substances which are not precipitated by tungstic solution as used in the Folin-Wu[2] procedure which follows:

A. Blood Filtrate
Into a flask place 2 cc. of oxalated blood
Add 16 cc. of N/12 sulfuric acid
Mix
Add 2 cc. of 10% sodium tungstate
Mix well
Filter through Whatman filter paper No. 40 (filtrate should be clear)

B. Same as Part B in Somogyi method

Method of yeast fermentation:[6, 7]

Dissolve a cake of commercial yeast in about 30 cc. of distilled water. Transfer to centrifuge tubes. Spin down (10 min.—3000 rpm). Pour off supernatent fluid. Wash this four times. Make up to 20 per cent solution with distilled water. Pour 10 cc. of this solution into 3 centrifuge tubes. Spin down. Pour off supernatent fluid. Add 6-10 cc. of Folin-Wu filtrate to one tube. Add 6-10 cc. of Somogyi filtrate to a second tube. Add distilled water to the third tube to serve as a blank. Stir each one with a glass stirring rod until the suspension is even. Set aside for 10-15 minutes at room temperature. Spin down.

Two cc. of the supernatent fluid from each centrifuge tube is placed in a Folin-Wu sugar tube. Two cc. of copper tartrate solution is added to each one. Proceed as in B above to determine the reducing substances left in the filtrates.

Note. If the yeast was sufficiently washed, the blank will read negative. If it does not read negative, it can be substituted for the blank already in the colorimeter, and the determinations can then be read on the colorimeter.

Table 1 lists four determinations in which a known amount of glucose was added to blood where the glucose content had been determined by both Folin-Wu and Somogyi methods. Recoverability was within 3 milligrams per cent using the Somogyi technique.

TABLE 1 (see text for explanation)

Folin-Wu	*Somogyi*	*Somogyi with 100 mg.% glucose added*
133 mg.%	107 mg.%	208 mg.%
208	183	280
305	290	390
308	248	345

Table 2 indicates parallel blood sugar determinations using both methods and the readings for both methods on the resultant filtrate after yeast fermentation had been carried out. It will be noted that all examples after fermentation read zero with the Somogyi procedure, with the exception of one 2 milligram reading. It is also readily apparent from the table that the artifact in the Folin-Wu procedure with regard to non-glucose reducing substances varies from 10 to 33 milligrams. Conversely, the Somogyi technique allows accurate prediction of the amount of non-glucose reducing substance since the original difference between the methods checks well with the amount left in the Folin-Wu filtrate after fermentation.

TABLE 2

Folin-Wu	*Somogyi*	*Folin-Wu after yeast fermentation*	*Somogyi after yeast*
103 mg.%	82 mg.%	18 mg.%	1 mg.%
212	192	19	0
315	280	33	0
200	188	10	2
224	193	27	0
128	107	19	0
118	86	26	0

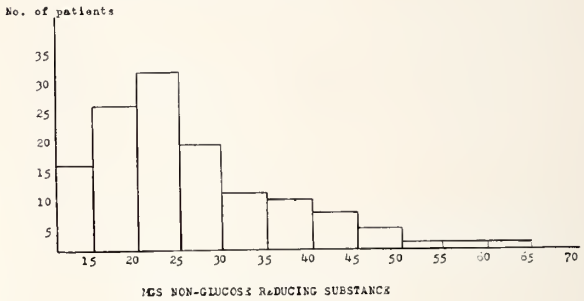
To check the reproducibility of the Somogyi method, we determined results in duplicate on 10 patients. The readings were never more than 3 milligrams apart.

Table 3 lists 96 blood sugar determinations using the two methods in parallel, and it can be seen that the lowest amount of non-glucose reducing substance was 10 milligrams and the greatest 63 milligrams per cent.

TABLE 3

Folin-Wu	*Somogyi*	*Difference*
110 mg.%	87 mg.%	23 mg.
133	115	18
345	305	40
92	82	10
230	202	28
103	73	30
110	79	31
148	124	24
92	70	18
268	230	38
224	193	31
128	96	32
140	109	31
137	110	27
165	137	28
100	64	36
240	224	16
165	144	21
212	188	24
103	75	28
188	170	18
255	210	45
103	75	28
105	85	30
107	55	42
107	60	47
323	260	63
208	150	58
212	155	57
148	98	50
140	90	50
98	55	37
212	150	62
110	65	45
110	75	35
103	52	51
305	290	15
280	248	32
202	160	42
128	92	36
255	235	20
212	198	20
110	79	21
92	64	28
103	79	23
88	60	28
165	150	15
133	107	26
107	90	17
117	92	25
208	183	25
103	70	33
305	290	15
280	248	32
260	240	20
167	154	13
100	79	21
137	107	30
275	260	15
212	192	20
315	280	35
212	190	22
112	92	20
153	140	13
200	188	12
110	85	25
280	268	12
153	135	18
200	170	30
107	80	27
100	75	25
96	82	14
110	82	28
153	117	36
113	96	17
103	83	20
110	92	18
335	315	20
197	181	16
110	88	22
100	85	15
113	100	13
188	165	22
103	82	21
335	323	12
113	88	25
198	183	15
175	153	22
224	193	32
128	107	21
100	79	21
100	75	25
185	130	55
110	87	23
116	86	30
148	124	24

Graph 1 reveals 131 patients with amounts of non-glucose reducing substance increasing in increments of 5 mg. The vertical bars indicate the number of patients, the horizontal bars being the milligrams of non-glucose reducing substances. A glance will show that there are 16 patients with a discrepancy between the two methods of from 10 to 15 mg., whereas 26 revealed a difference of 15-20 mg., 32 a difference of 20-25 mg., 18 a difference of 25-30 mg., 11 a difference of 30-35 mg., et cetera.



GRAPH 1

Table 4 lists the fasting blood sugar of 7 diabetics with blood sugars taken over a year's period of time. We were interested to discover if, for a given period, the quantity of non-glucose reducing substance stays approximately the same. It is evident from this table that such is not the case, there being as much as 51 milligrams difference in one patient from time to time. We have not determined whether the non-glucose reducing substances stay the same over any one period of time for a given level of blood sugar.

TABLE 4 (See Text)

Patient	*Folin-Wu*	*Somogyi*	*Difference*
Mrs. L	345 mg.%	305 mg.%	40 mg.
	212	188	24
	323	260	63
	305	290	15
	305	290	15
	275	260	15
	280	268	12
Mr. N	230	202	28
	140	90	50
	202	160	42
	224	193	32
Mrs. S	110	79	31
	212	192	20
Mrs. G	153	117	36
	268	230	38
	255	210	45
	167	154	13
	280	248	32
	280	248	32
	200	170	30
Mrs. G	224	193	31
	198	183	15
	212	155	57
	208	183	25
	200	188	12
Mrs. D	140	109	31
	110	75	35
	133	107	26
	113	100	13
	148	124	24
Mr. M	116	86	30
	110	88	22
	112	92	20
	92	64	28

Table 5 gives the results of glucose tolerance tests with parallel determinations and indicates the milligrams of non-glucose reducing substances found in 7 patients, only 1 of whom was diabetic.

TABLE 5

Patient		*Folin-Wu*	*Somogyi*	*Difference*
Mrs. S	Fasting	103 mg.%	75 mg.%	28 mg.
	1 hr.	96	85	11
	2 hr.	79	55	24
	3 hr.	85	70	15
	4 hr.	92	73	19
Mr. B	Fasting	92	47	45
	1 hr.	110	52	45
	3 hr.	60	35	25
	4 hr.	88	50	38
Mr. D	Fasting	101	56	45
	½ hr.	140	100	40
	1 hr.	124	75	49
	2 hr.	96	72	24
	3 hr.	60	39	21
	4 hr.	75	47	28
Miss. McC	Fasting	110	73	37
	1 hr.	193	144	49
	2 hr.	183	165	49
	3 hr.	128	104	24
	4 hr.	137	93	44
Mr. S	Fasting	117	82	35
	1 hr.	133	114	19
	2 hr.	82	58	24
	3 hr.	117	93	24
	4 hr.	100	70	30
Mrs. F	Fasting	103	82	21
	1 hr.	160	140	20
	2 hr.	102	82	20
	3 hr.	86	68	18
	4 hr.	85	66	19
Mrs. H	Fasting	101	76	25
	1 hr.	120	100	20
	2 hr.	107	85	22
	3 hr.	90	70	20
	4 hr.	88	70	18

COMMENTS

To discover if the above techniques are practical, we have carried them out in our office laboratory, and it has become apparent that there is no more labor involved in the Somogyi technique than in the more-commonly used and, we believe, out-moded Folin-Wu procedure. The criteria mentioned at the beginning of this paper, that is, method of determination, reproducibility, determination of true blood sugar, seem to be fulfilled.

It should be stated that we have not observed the Folin-Wu procedure to label a patient falsely diabetic, remembering that the standard for the Folin-Wu method is a fasting blood sugar below 120 mg. per cent (or at the end of the second hour of a glucose tolerance test), whereas that for the Somogyi technique is similarly below 100 mg. per cent. Nevertheless, it seems to us entirely possible that through the years, with the discrepancy between the two methods being as variable and as high as it occasionally develops, such an error is ultimately inevitable.

One might also comment on the current dispute between those who advocate very strict regulation of the diabetic and those doctors who teach a freer technique in that with as much as 60 mg. of artifact occurring in the Folin-Wu method, this being the method almost universally used at present, normoglycemic fasting blood sugar is actually dangerously low in some patients.

SUMMARY AND CONCLUSIONS

In summary, we believe that the Somogyi method should be adopted for the routine determination of blood sugars. It is conveniently adaptable to the average office and hospital and considerably more informative than the Folin-Wu procedure now in common use. We have found that the reducing substance determined by the former procedure is entirely removed by fermentation with yeast, and that from 10 to 63 mg. of reducing substance may remain after glucose is removed by fermentation. Further, this amount of reducing substance is reported as glucose in the Folin-Wu technique.

Periodic fasting blood sugar determinations in diabetics have revealed a variable and inconstant amount of non-glucose reducing substance ranging from 12 to 57 milligrams per cent. Also glucose tolerance tests reveal that during a four hour determination there may be as much as 49 milligrams per cent variation between glucose and total reducing substance in the blood.

BIBLIOGRAPHY

1. Mosenthal, H. O., and Barry, E.: Criteria for the interpretation of normal glucose tolerance tests, Proc. Am. Diab. Assn. 9:277, 1949.
2. Folin, Otto, and Wu, Hsien: A simplified and improved method for determination of sugar, J. Bio. Chem. 41:367, 1920.
3. Fiorentino, M., and Giannettasio, G.: Photometric blood sugar determination by the Folin-Wu method. J. Lab. and Clinical Med. 25:866, 1940.
4. Somogyi, Michael: A method for the preparation of blood filtrates for the determination of sugar, J. Bio. Chem. 86:655, 1930.
5. Somogyi, Michael: Reducing non-sugars and true sugar in human blood, J. Bio. Chem. 75:33, 1927.
6. Nelson, Norton: A photometric adaptation of the Somogyi method for the determination of glucose, J. Bio. Chem. 153:375, 1944.
7. Somogyi, Michael: The distribution of sugar in normal human blood, J. Bio. Chem. 78:117, 1928.

Science seeks truth and discovers rightness. Religion seeks righteousness and discovers truth. Both have acquired knowledge of creative and destructive ways, and both point the same way of right living. —*William G. Patten*

PRESIDENT'S PAGE

Dear Doctor:

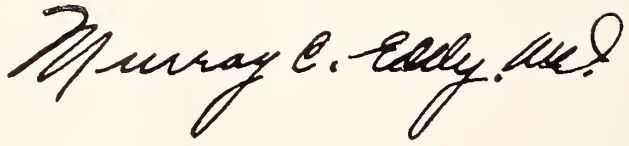
Yesterday, today and tomorrow converge in this era of speed. The dreams of yesterday are distorted in the frenzy of today and are thrust into tomorrow's reality. Medicine illustrates this platitude both vertically and in a lateral plane, both through its scientific achievement and its sociological evolution.

Yesterday, in the dawn of medical discovery the physician found new means for preventing disease and for arresting its progress. He traveled long weary hours in the darkness and through mud to minister to those in need, but he walked a free man unafraid of his future, unfettered by governmental regulations and controls.

Today his vertical progress is expected and demanded by the public. What was considered a miracle cure a short time ago is now taken for granted. But with this opportunity for increasingly greater service comes a strangling lateral control. The ideal of individual achievement is being crushed out in favor of mediocrity, in favor of the average. Today conformity is the rule and society is the king.

Tomorrow those influences will be united and woven into a single pattern. Its effect will be determined by what we do today, in our professional career as well as in our citizenship. If ever we had a need for clear thinking and decisive action in the preparation of a future that day is now. As physicians we perhaps have an understanding of our duties but as citizens we must waken to a new responsibility. One, among the many opportunities in this direction grows out of our right to vote. May we not neglect this expression of good citizenship in November.

Yours,



EDITORIAL COMMENT

A History of the Society

On February 10, 1959 The Kansas Medical Society will become one-hundred years of age. This notable date is just four and one-half years away and plans are now being formed for a fitting commemoration of this event.

This Society was chartered by the Territorial Legislature before Kansas became a state. Even at that time, the hardy immigrant pioneer physicians who incorporated the Society envisioned an organization that would improve the practice of medicine for the purpose of better serving the people of this new land.

Early transactions are reviewed with a smile where an occasional enthusiast tells his colleagues that the end of all medical discovery is at hand, or in such homely soft spots where everyone's dues were forgiven because the secretary lost the ledger, or in the record of spirited differences of professional opinion as for instance, a discussion of the scientific possibilities of mesmerism.

Not funny at all are the discussions of needs for higher standards. From the very earliest meetings appear pleas for a license law whereby physicians would be required to show some educational qualifications before being permitted to practice in this state. Nearly one-hundred years ago they appeared before the legislature asking for quarantine laws, for vital statistics records and for the power to enforce sanitary measures in an effort to avert the ever threatening epidemics of cholera, smallpox and diphtheria. They begged first the Board of Regents and then the legislature repeatedly for a chair of medicine at Kansas University.

They worked then as does the Society today for such improvements as will provide for a healthier population. In the early records they expressed pride for having attained a remarkable life expectancy of 40 years. By 1900 they were rejoicing that this had been raised to 49 years just as the Society today expresses pleasure that this figure stands at 69 years. And then, just as in the case today, they were looking ahead toward greater improvements in the future.

This Society has grown great because of its uncompromising insistence upon maintaining the highest ideals of medicine. Its unwavering acceptance of the responsibility to be of service to the public has been a continuing challenge. This forward looking view has kept the Society active and has kept it young which, be it hoped devoutly, shall always be its course.

However, a backward glance is not completely in vain, especially upon the approach of a centennial. Here the tragic revelation becomes apparent that history has not been preserved. The forefathers of this Society were so engrossed with affairs of the day at hand, that the past has all but been obliterated.

Gone are priceless experiences of how medicine was practiced in the past. Gone are the biographies of the pioneers in Kansas medicine. Departed from memory are the circumstances upon which great decisions were made.

As the centennial approaches the Committee on History hopes to prepare a record of The Kansas Medical Society. They most hopefully request assistance in gathering data for this project. Every recollection of past experience will be of interest. The biography of an early member will be appreciated. Samples of early instruments, the story of the use of drugs in the 19th century, the organization of county societies, early text books will all be of value.

This committee urgently requests any member who has access to any information, however slight, concerning medicine in the early days of this state, to send it to The Kansas Medical Society, 315 West Fourth Street, Topeka, Kansas. Similarly if anyone can suggest where such material may be located, that information will be of value. It would be most appreciated if a wealth of data could be accumulated now so the history can be prepared by 1959, in time for the centennial. Your personal help can make this project successful.

Federal Health Programs

PART III

This installment completes a review of federal participation in health programs. As stated in Part I, see the July, 1954, JOURNAL, the federal government will have spent during this fiscal year more than one and three-quarters of a billion dollars in some sixty separate agencies dealing with health. The previous sections discussed some of these programs and the remainder appear below.

The Department of Labor expected to spend during the fiscal year closing June 30, 1954, $8,960,000, mostly in two fields. The Bureau of Employees Compensation accounts for about $8,500,000. Its service is roughly to federal employees what Workmen's Compensation is to the states. There are more than two million persons covered and benefits include medical, hospital and burial expenses. The second is the Health and Safety Program with a budget of just under a half million. This includes such programs as for re-employment of the physically handicapped and the promotion of industrial safety.

The Foreign Operations Administration spent $24,500,000 for health out of a total budget of more than $118,000,000 to aid backward countries. Last

year the health fund was spent as follows: Six million for Latin America, seven million for the Near East and Africa, and eleven million for South and Southeast Asia, to mention only the major contributions.

The Department of Interior spent $27,258,600 for health under three divisions. The Bureau of Indian Affairs received $21,400,000 to operate 59 Indian hospitals, for contract health services and including $551,687 for administration and salaries. The Bureau of Mines spent $5,060,000 of which $3,700,000 was for mine inspection, $850,000 for investigations of accidents and rescue work, and $510,000 for explosives testing and research. The third is the Alaskan Mental Health Program with a budget of $798,600.

The Department of Commerce spent $621,000 for health of which half is used by the Bureau of Standards to test such things as hearing aid batteries, substitutes for blood plasma, heart pumps and artificial kidneys, etc. The other half goes to the Civil Aeronautics Administration to carry out the medical portion of this authority's flight safety program. Fourteen medical officers supervise the annual physical examinations of commercial and private pilots, a total of some 160,000 who pay for their examinations. This does not come from federal funds.

The Department of the Treasury spent $2,790,000 on narcotic control, about 90 per cent of which went for salaries. The Department of Justice spent $1,326,000 under the Bureau of Prisons for medical and dental care for some 18,000 prisoners in 27 federal penal institutions.

There follows a listing of independent offices not directly attached to any other bureau or department. The National Science Foundation used $8,000,000 of which half was used to correlate research and services of other agencies and half for research grants.

The Federal Civil Service Administration had $26,650,000 earmarked for health and medical stockpiling, including $250,000 for salaries and travel.

The Atomic Energy Commission spent $26,565,000 as follows: $10,376,700 for research on the effects of radiation; $8,036,300 for general medical research including radioisotopes; $4,788,900 for biophysics; $2,833,100 for cancer research; and $530,000 for fellowships and special training. The above is just medical work out of a total A.E.C. budget of $1,057,781,000.

The Health Resources Advisory Committee used $91,000, and the National Advisory Committee to Selective Service spent $265,000.

Medical Care in the Panama Canal Zone cost $5,448,600 for the operation of four hospitals with a total of 1,400 beds, six outpatient medical clinics, two dental clinics, quarantine service, insect control programs and others.

Medical work of the Federal Trade Commission accounts for one-fourth of its total budget or about one-million dollars to administer food, drug and cosmetic controls.

The Commission on Intergovernmental Relations operates with a budget of $500,000 to study the services of all the agencies mentioned in this report. This 25-man commission has until September 1 of this year to complete its fact finding program.

The Commission of the Executive Branch of the Government consists of twelve members under Herbert Hoover. It is interested in promoting efficiency and in reducing government competition to free enterprise. This commission has $500,000 and is required to complete its survey by May 31, 1955.

Federal Employees Health Programs spent $6,000,000 last year for health services to keep federal employees on the job. Most of this money was spent for pre-employment examinations, for clinics to treat on-the-job illnesses and accidents, and for such care, as for example, the giving of insulin, if such is recommended by the family physician.

This three-part review was taken from a report issued by the American Medical Association's Washington Office in the interest of providing information to the medical profession upon federal participation in health programs. A considerable portion of these services is performed within the states and often with state financial support. This series will be continued in future issues to show where Kansas receives federal support and how such funds are spent.

A.M.A. Annual Session

Many physicians have read the proceedings of the recent meeting of the American Medical Association which was held in San Francisco in June of this year. A number of Kansas physicians journeyed to the West Coast to take part in the meeting. Some presented scientific exhibits and others read a paper. Delegates representing The Kansas Medical Society fulfilled their responsibilities and still other physicians took advantage of the clinical sessions and the many other opportunities afforded at that meeting.

Noticeably absent during the business session of the House of Delegates was the usual confusion and debate which generally arises over the many resolutions presented. Although some seventy-five resolutions were acted upon, various committees had spent considerable time throughout the year studying the problems and reporting at the meeting. Specific instructions on many of the resolutions were given by state societies to their delegates prior to the meeting

which was another factor contributing to the smooth functioning of the meeting. For those who have not had access to information on the meeting, the following is a condensed version of some of the important action taken by the A.M.A.

Several resolutions by various state medical societies were introduced regarding osteopathy and medicine. The Committee of the Board of Trustees which submitted a report to the A.M.A. House of Delegates in June, 1953, suggested at that time that osteopathy be removed from the "cultist" classification and that doctors of medicine be permitted to teach in osteopathic schools. The principle criticism of this report was that the committee had to rely on indirect evidence concerning the nature and scope of education in osteopathic schools. The committee reported at the recent meeting that it had proposed to The American Osteopathic Association that a special committee be established to visit schools of osteopathy to determine the quality of medical education provided. The A.M.A. approved of this action.

The House of Delegates reconfirmed their stand on "fee splitting, rebating or payment of commissions in any form whatever." This problem has been studied the past two years by the Committee on Constitution and By-Laws of the A.M.A. and after a lengthy discussion in reference committee, their report was adopted.

The stand of the A.M.A. regarding veterans medical and hospital care remained unchanged. The A.M.A. will continue to seek legislation which would limit medical care in Veterans Administration and other hospitals to:

A. Veterans with peacetime or wartime service whose disabilities or diseases are service-incurred or aggravated.

B. Within limits of existing facilities to veterans with wartime service suffering from tuberculosis or psychiatric neurological disorders of nonservice connected origin, who are unable to defray the expense of necessary hospitalization.

It was again suggested that veterans with nonservice connected disabilities who are unable to defray expense for such care can receive attention from local personnel and facilities.

Smaller hospitals (under 100 beds) will receive attention during the coming year by the A.M.A. The Council on Medical Education and Hospitals was asked to study and set up a system of standardization for smaller hospitals. Those in small hospitals find it difficult to maintain the standards set by the Joint Commission on the Accreditation of Hospitals.

Dr. Edward J. McCormick, immediate past president of the A.M.A. urged physicians to adopt average fee schedules on an area or regional basis to overcome a large number of complaints by patients in regard to fees. This would also enable the patient to determine the ultimate cost of the medical service he seeks.

The seal of acceptance of voluntary health insurance plans will be discontinued by the A.M.A. Because of varying state laws, it is feared that the use of the seal might give the public an erroneous impression. It is suggested that any person purchasing or recommending any health insurance plan might examine the plan in the light of A.M.A. standards and principles which will be available as a guide.

Dr. Walter J. Martin of Norfolk, Virginia, was installed as President of the A.M.A. and Dr. Elmer Hess of Erie, Pennsylvania, was unanimously elected to the office of President-Elect at the final meeting of the House of Delegates on June 24, 1954.

BOOK REVIEW

Endemic Goiter, The Adaptation of Man to Iodine Deficiency, by John B. Stanbury, M.D., Gordon L. Brownell, Ph.D., Douglas S. Riggs, M.D., Hector Perinetti, M.D., Juan Itoiz, Ph.D., Erique B. Del Castillo, M.D., Harvard University Press, Cambridge, Massachusetts, 206 pp., 53 figures, $4.00.

This is the report by a distinguished group of investigators on their physiological studies of the iodine starved thyroid gland as found in Mendoza Province of Argentina, where it has been reported that four fifths of the men and women of the province have visible tumors of the neck. The method of study included the use of tracer doses of radioactive iodine, with the measurement of uptake and excretion. The effect on the distribution of iodine in the body from administering additional iodine, desiccated thyroid, methimazole, and thyrotropin, was also studied. Mathematical calculations based on several assumptions gave results reasonably close to the observed values. In a relatively short space one is acquainted with the frontiers of the goiter problem.

The iodine content of the drinking water in Mendoza was found to be 2.1, 3.0, and 2.0 micrograms per liter, which was compared with 27.8 and 18.2 micrograms per liter for tap water in Boston on two determinations. No reference is made to the book by McClendon, *Iodine and the Incidence of Goiter*. Since these studies were completed the Argentine Government has made the sale of iodized salt compulsory. Although the iodine content of the drinking water in much of the midwest is reported to be even less than that found in Mendoza, we still have our choice of plain or iodized salt at our grocery. If we choose the iodized, one gram of the salt per day contains enough iodine to prevent goiter.—*K.Y.*

Supplement to Roster

In June 1953, this Society with the Kansas State Board of Medical Registration and Examination published a Roster of Kansas Physicians. During the interval since that date, numerous changes have occurred to render it comparatively obsolete, so elsewhere in this JOURNAL may be found a list of names which when used with the previously published Roster will again make it usable.

Certain explanations may be necessary for an understanding of how this supplement might be used. This new listing includes members only. Information on nonmembers would be obtained only with some difficulty so no attempt has been made to correct changes as they affect names which appear in lower case print in the Roster.

1. Members—To Be Added, lists names of physicians, now members of this Society who have joined since June 1953. They may have recently moved to this state, or may have been previously listed in lower case type.

2. Members—Address Changes, lists those members (in capital letters) from the 1953 Roster whose present address differs from the previous printing. Since office addresses are given only for physicians in the larger cities, these are listed if the office address is changed. Otherwise, in all the smaller towns the name appears only if the member moved to a different community from that listed in 1953.

3. The section headed "Alphabetized by Towns" merely regroups all the names appearing under "Members to Be Added" and "Members—Address Changes" according to their present location. It should be recalled that this is not necessarily a complete listing of physicians in any named community. This records only the changes that have occurred since the last Roster was published and should be used in connection with it.

4. Deletions is a record of members whose names may be taken off the previous Roster. They are deceased or have moved out of the state and in one or two instances may still be in the previously listed location. However, they are not members of The Kansas Medical Society at the present time and for the purposes of the supplement have therefore been removed from the listing.

The Supplement, therefore, is not a new Roster, but merely an attempt to bring the other up-to-date. It lists only those changes that affect members and will become useful only in connection with the previous publication. In 1953 each member received a copy. Some are still available and may be purchased with the supplement at a cost of $1.00. Should this be desired, they may be obtained through The Kansas Medical Society, 315 West Fourth Street, Topeka, Kansas.

New Members

Aronson, Gerald ..... Columbia, S. C.
Ashley, E. Vernon ..... Bushton
Atwood, M. Dale ..... Kinsley
Baeke, John O. ..... Overland Park
Bayles, Hugh G. ..... Fredonia
Bayles, Spencer ..... Kansas City
Benkelman, Ward E. ..... Polson, Montana
Bill, Robert O. ..... Topeka
Birdsell, John E. ..... Topeka
Bly, Chauncey G. ..... Kansas City
Bodmer, Robert A. ..... Coldwater
Brooker, Robert M. ..... Topeka
Brooks, R. D. ..... Madison
Buddrus, David J. ..... Prairie Village
Budetti, Joseph A. ..... Wichita
Burgess, Arthur P. ..... Oswego
Calderwood, Barbara ..... Phillipsburg
Christensen, Marion D. ..... Kiowa
Custer, Harry R. ..... Colby
Delaney, W. M. ..... Topeka
Diacon, James L. ..... Wellington
Diehl, Antoni M. ..... Kansas City
Diehl, Leonard R. ..... Osborne
Dittemore, James H. ..... Belleville
Dobratz, Robert A. ..... Chetopa
Dreyer, Richard ..... Salina
Ens, Peter D. ..... Hillsboro
Evans, Mary E. ..... Kansas City
Fatland, John L. ..... Council Grove
Firminger, Harlan I. ..... Kansas City
Fitzpatrick, Martin J. ..... Kansas City
Fowler, W. L. ..... Concordia
Frederickson, Evan L. ..... Kansas City
Gendel, Evalyn S. ..... Auburn
Griffith, John K. ..... Effingham
Guggenheim, Paul ..... Topeka
Gunter, Carl G. ..... Quinter
Haley, Jack A. ..... Hanover
Hamm, Orville L. ..... Gardner
Hassard, George H. ..... Sedan
Heasty, Alfred ..... Conway Springs
Heffner, Edward A. ..... Hutchinson
Hege, John H. ..... Sunflower
Heisterman, Herman W. ..... Quinter
Hoff, Donald E. ..... Wichita
Hooker, Lyle T. ..... Osawatomie
Hull, Norman E. ..... Hays
Hutchinson, Glen C. ..... Hays
Ivy, H. B. ..... Meridian, Mississippi
Jackson, George W. ..... Topeka
Jastram, R. M. ..... Minneapolis, Minnesota
Jenkins, Neal M. ..... Salina
Ketterman, Grace H. ..... Kansas City
Kiser, David M. ..... Overland Park
Kittle, C. Frederick ..... Kansas City
Kramer, Lee F. ..... Cherryvale

Laaser, William E. ..... Kansas City
Laurens, Henry ..... Salina
Lessenden, C. M., Jr. ..... Topeka
Lothman, T. L. ..... Enterprise
Lowry, Ray F. ..... El Dorado
Lund, Leonard C. ..... Osawatomie
McCann, William ..... Olathe
McClymond, Robert C. ..... Walton
McGonigle, Lester E. ..... El Dorado
McGovern, James L. ..... Wellington
McGuire, George E. ..... Wichita
McGuire, John W. ..... Neodesha
Madison, Willard A. R. ..... Nortonville
Magee, Charles Raymond ..... Marion
Mastio, George J. ..... Ulysses
Miller, William Robert ..... Axtell
Mueller, Adolph Reginald ..... Leavenworth
Neis, Harry B. ..... Olathe
Nesselrode, John H. ..... Kansas City
Nutting, Harry C. ..... Emporia
Olson, Erwin T. ..... Newton
Orrison, William W. ..... Minneola
Osborne, J. O. ..... Ness City
Owensby, Lindell C. ..... Concordia
Page, Vale ..... Plainville
Parker, David Findley ..... Tonganoxie
Parker, Joseph W. ..... Burlington
Pearce, Lunetta M. ..... Sunflower
Plowman, Carl W. ..... Jewell
Pogson, George W. ..... Pittsburg
Powell, Charles ..... Columbus
Preheim, Delbert Victor ..... Newton
Rhoades, Gordon Howard ..... Halstead
Rice, David R. ..... Moundridge
Ridlon, Albert M. ..... South Whitley, Indiana
Rottersman, William ..... Topeka
Rowlett, Jack G. ..... Paola
Schraer, Paul ..... Concordia
Schwartz, Eugene ..... Dodge City
Schweinfurth, James P. ..... Wichita
Siler, Eugene T. ..... Hays
Smith, James D. ..... Satanta
Spanos, William J. ..... Kansas City
Stanley, Rex C. ..... Paola
Struxness, Erling B. ..... Hutchinson
Sullivan, Henry B., Jr. ..... Shawnee
Sweeney, John E. ..... Topeka
Tarnower, William ..... Topeka
Taylor, James A. ..... Muncie, Indiana
Terrill, Robert S. ..... Topeka
Thompson, Victor J., Jr. ..... Topeka
Throckmorton, Marion ..... Halstead
Tikellis, I. J. ..... Lawrence
Todd, Richard N. ..... Kansas City, Missouri
Tozer, Richard C. ..... Topeka
Treger, Donald M. ..... Bremerton, Washington
Vogt, William L. ..... Champaign, Illinois
Voorhees, Carroll D. ..... Leavenworth
Weldon, Thomas A. ..... Perry
Wenner, Herbert A. ..... Kansas City
Williams, Donald L. ..... Garden City
Williamson, Paul ..... Osawatomie
Wilson, Vernon E. ..... Kansas City
Wood, Robert G. ..... Coffeyville
Woods, Hugh J. ..... Smith Center

Members—Address Changes

Aleshire, Irma ..... Iowa City, Iowa
Artman, John C. ..... Kearney, Nebraska
Bair, Albert E. ..... Wichita
Bantleon, V. H. ..... Kansas City
Belisle, R. C. ..... Hays
Bell, Bertha E. ..... Ottawa
Bice, Francis J. ..... Wakeeney
Bishop, Francis E. ..... Massapequa, New York
Blevins, Walter E. ..... Liberal
Bonfanti, Albert ..... Junction City
Bowen, Murray ..... Bethesda, Maryland
Brownlee, William ..... Mission
Calkins, Larry L. ..... Kansas City
Campbell, Charles A. ..... Phillipsburg
Case, Laurel G. ..... Wichita
Claypool, J. Gordon ..... Narberth, Pennsylvania
Coffey, Roy B. ..... New York, New York
Coffman, James H. ..... El Dorado
Coyle, John F. ..... Panama City, Florida
Critchfield, T. W. ..... Seattle, Washington
Crockett, C. A. ..... Arlington, Virginia
Demand, J. W. ..... Long Beach, California
Dimond, E. Gray ..... Overland Park
Dunagin, Jack A. ..... Topeka
Edmiston, R. H. ..... Americus
Edwards, E. M. ..... Mt. Rainier, Maryland
Edwards, R. W. ..... West Los Angeles, California
Ehrlich, Karl A. ..... Oakland, California
Enna, Carl D. ..... Kansas City, Missouri
Ewing, T. D. ..... Larned
Fairchild, John P. ..... Fort Sam Houston, Texas
Fairchild, Robert C. ..... Mission
Fall, N. B. ..... Colorado Springs, Colorado
Floyd, Glen ..... La Jolla, California
Friesen, Florence ..... Hesston
Funk, Edward D. ..... Prairie Village
Garst, R. J. ..... Joplin, Missouri
Germann, Donald R. ..... Kansas City
Gianakon, Harry G. ..... Kansas City
Good, Wendell L. ..... Mission
Gootee, Joseph E. ..... Topeka
Grider, W. H. ..... Altadena, California
Gundle, Sigmund ..... Kansas City, Missouri
Gyarfas, W. J. ..... Duarte, California
Haffner, V. B. ..... Montevallo, Alabama
Hands, S. V. ..... Wichita
Harsha, W. N. ..... Oklahoma City, Oklahoma

Hays, A. D. ..... Baxter Springs
Helwig, G. F. ..... San Jose, California
Henry, Victor G., Jr. ..... Ft. Leavenworth
Hildyard, V. H. ..... Minneapolis, Minnesota
Hogan, T. W. ..... Terre Haute, Indiana
Hopp, Ralph L. ..... Ann Arbor, Michigan
Howard, Richard R. ..... Slidell, Louisiana
Hughes, Robert D. ..... Marysville
Huscher, Gladys ..... Concordia
Johnson, Charles H. ..... Mission
Johnson, L. D. ..... Kansas City, Missouri
King, E. R. ..... Cleveland, Ohio
Kridelbaugh, William W. .... Albuquerque, N. M.
Lance, Raymond W. ..... Arma
Laury, David G. ..... Olathe
Law, Findley ..... Bird City
Lockett, George R. ..... Concordia
McCoy, Charles T. ..... Hutchinson
Maier, Edwin R. ..... Rochester, Minnesota
Marrs, E. A. ..... Colorado Springs, Colorado
Martin, John N. ..... Chillicothe, Missouri
Maser, G. R. ..... Mission
Miller, Jack P. ..... Yakima, Washington
Mitchell, A. D. ..... Kansas City, Missouri
Montgomery, Charles ..... Roswell, New Mexico
Myers, Ben L. ..... Santa Rosa, California
Nevitt, R. R. ..... Iola
Nice, William ..... Topeka
Nickell, Wendell K. ..... San Angelo, Texas
Peck, J. H. A., Jr. ..... San Diego, California
Pederson, Arnold M. ..... Plainville
Peiper, Warren S. ..... Mitchell, S. D.
Pennington, Katherine ..... Baltimore, Maryland
Pottorf, Mildred M. ..... Wichita
Preston, Ralph R. ..... Topeka
Purinton, Lew W. ..... Norfolk, Virginia
Robison, Leonard J. ..... Irving, Texas
Roccaforte, Daniel S. ..... Salina
Scimeca, Salvatore A. ..... Wichita
Selzer, Donald W. ..... Aitkin, Minnesota
Sirridge, Marjorie S. ..... Fort Riley
Sirridge, William T. ..... Fort Riley
Smith, Carl M. ..... Clermont, Florida
Snyder, Z. H. ..... Hays
Stacey, Harley ..... Oklahoma City, Okla.
Sterrett, Preston ..... Humansville, Missouri
Tetzlaff, Fred M. ..... San Francisco, California
True, Otis H. ..... Kansas City, Missouri
Votn, Harold W. ..... Larson AFB, Washington
Walters, Orville S. ..... Topeka
Wartman, Calvin ..... Bremerton, Washington
Weber, Clarence J. ..... Salina
Weed, Randal ..... St. Joseph, Missouri
Weeks, Vernon L. ..... Fargo, North Dakota
Wellman, Garland O. ..... Texas City, Texas
White, R. Burnley ..... Winfield
Wolff, Frederick P. ..... Pratt

Listed By Cities

AMERICUS

Edmiston, R. H.

ARMA

Lance, Raymond W.

AUBURN

Gendel, Evalyn S.

AXTELL

Miller, William Robert

BAXTER SPRINGS

Hays, A. D.

BELLEVILLE

Dittemore, James H.

BIRD CITY

Law, Findley

BURLINGTON

Parker, Joseph W.

BUSHTON

Ashley, E. Vernon

CHERRYVALE

Kramer, Lee F.

CHETOPA

Dobratz, Robert A.

COFFEYVILLE

Wood, Robert G.

COLBY

Custer, Harry R.

COLDWATER

Bodmer, Robert A.

COLUMBUS

Powell, Charles

CONCORDIA

Fowler, W. L.
Huscher, Gladys
Lockett, George R.
Owensby, Lindell C.
Schraer, Paul

CONWAY SPRINGS

Heasty, Alfred

COUNCIL GROVE

Fatland, John L.

DODGE CITY

Schwartz, Eugene

EFFINGHAM

Griffith, John K.

EL DORADO

Coffman, James H.
Lowry, Ray F.
McGonigle, Lester E.

EMPORIA

Nutting, Harry C.

ENTERPRISE

Lothman, T. L.

FORT LEAVENWORTH

Henry, Victor G., Jr.

FORT RILEY

Sirridge, Marjorie S.
Sirridge, William T.

FREDONIA

Bayles, Hugh G.

GARDEN CITY

Williams, Donald L.

GARDNER

Hamm, Orville L.

HALSTEAD

Rhoades, Gordon Howard
Throckmorton, Marion

HANOVER

Haley, Jack A.

HAYS

Belisle, R. C.
Hull, Norman E.
Hutchinson, Glen C.
Siler, Eugene T.
Snyder, Z. H.

HESSTON

Friesen, Florence

HILLSBORO

Ens, Peter D.

HUTCHINSON

Heffner, Edward A.
McCoy, Charles T.
Struxness, Erling B.

IOLA

Nevitt, R. R.

JEWELL

Plowman, Carl W.

JUNCTION CITY

Bonfanti, Albert

KANSAS CITY, KANSAS

Bantleon, V. H.
14 South 14th Street
Bayles, Spencer
U. of K. Medical Center
Bly, Chauncey G.
U. of K. Medical Center
Calkins, Larry L.
211 Huron Building
Cooper, Jack R.
New Brotherhood Bldg.
Diehl, Antoni M.
U. of K. Medical Center
Evans, Mary E.
U. of K. Medical Center
Firminger, Harlan I.
U. of K. Medical Center
Fitzpatrick, Martin J.
U. of K. Medical Center
Frederickson, Evan L.
U. of K. Medical Center
Germann, Donald R.
U. of K. Medical Center
Gianakon, Harry G.
16 North Thorpe
Harms, Albert C.
2121 Minnesota Ave.
Ketterman, Grace H.
619 Ann
Kittle, C. Frederick
U. of K. Medical Center
Knight, D. K.
640 Minnesota Ave.
Laaser, William E.
2501 North 45th St.
Lee, James Grant, Jr.
4367 Troup Ave.
Lowman, R. C.
816 North 9th
Nesselrode, John H.
921 Hillcrest Road
Spanos, William J.
U. of K. Medical Center
Strobach, Rolla L.
1811 Quindaro Blvd.
Wenner, Herbert A.
U. of K. Medical Center

Wilson, Vernon E.
U. of K. Medical Center

KINSLEY

Atwood, M. Dale

KIOWA

Christensen, Marion D.

LARNED

Ewing, T. D.

LAWRENCE

Tikellis, I. J.

LEAVENWORTH

Mueller, Adolph Reginald
Voorhees, Carroll D.

LIBERAL

Blevins, Walter E.

MADISON

Brooks, R. D.

MARION

Magee, Charles Raymond

MARYSVILLE

Hughes, Robert D.

MINNEOLA

Orrison, William W.

MISSION

Brownlee, William
Fairchild, Robert C.
Good, Wendell L.
Johnson, Charles H.
Maser, G. R.

MOUNDRIDGE

Rice, David R.

NEODESHA

McGuire, John W.

NESS CITY

Osborne, J. O.

NEWTON

Olson, Erwin T.
Preheim, Delbert Victor

NORTONVILLE

Madison, Willard A. R.

OLATHE

Laury, David G.
McCann, William
Neis, Harry B.

OSAWATOMIE

Hooker, Lyle T.
Lund, Leonard C.
Williamson, Paul

OSBORNE

Diehl, Leonard R.

OSWEGO

Burgess, Arthur P.

OTTAWA

Bell, Bertha E.

OVERLAND PARK

Baeke, John O.
Dimond, E. Grey
Kiser, David M.

PAOLA

Rowlett, Jack G.
Stanley, Rex C.

PERRY

Weldon, Thomas A.

PHILLIPSBURG

Calderwood, Barbara
Campbell, Charles A.

PITTSBURG

Pogson, George W.

PLAINVILLE

Page, Vale
Pederson, Arnold M.

PRAIRIE VILLAGE

Buddrus, David J.
Funk, Edward D.

PRATT

Wolff, Frederick P.

QUINTER

Gunter, Carl G.
Heisterman, Herman W.

SALINA

Dreyer, Richard
Jenkins, Neal M.
Laurens, Henry
Roccaforte, Daniel S.
Weber, Clarence J.

SATANTA

Smith, James D.

SEDAN

Hassard, George H.

SHAWNEE

Sullivan, Henry B., Jr.

SMITH CENTER

Woods, Hugh J.

SUNFLOWER

Hege, John H.
Pearce, Lunetta M.

TONGANOXIE

Parker, David Findley

TOPEKA

Beverly, W. B.
2410 Granthurst
Bill, Robert O.
921 Topeka Ave.
Birdsell, John E.
Santa Fe Hospital
Brooker, Robert M.
420 Garlinghouse Bldg.
Delaney, W. M.
Santa Fe Hospital
Dunagin, Jack A.
1266 Collins
Fink, Arthur A.
1320 Pembroke Lane
Gootee, Joseph E.
3401 West 10th
Guggenheim, Paul
Winter VA Hospital
Jackson, George W.
801 Harrison
Johnson, James S.
825 Clay
Lessenden, C. M., Jr.
723 Mills Building
McNaughton, James H.
1319 West 16th
Morris, H. T.
1401 West 10th
Morris, M. D.
1401 West 10th
Nice, William
1425 West 10th
Preston, Ralph R.
1733 Pembroke Lane
Rottersman, William
3617 West 6th Street
Schwartz, Lloyd G.
839 North Kansas Ave.
Sweeney, John E.
10th & Fillmore
Tarnower, William
Topeka State Hospital
Terrill, Robert S.
Santa Fe Hospital
Thompson, Victor J., Jr.
330 Kansas Avenue
Tozer, Richard C.
3617 West Sixth
Walters, Orville S.
1272 Medford Ave.

ULYSSES

Mastio, George J.

WAKEENEY

Bice, Francis J.

WALTON

McClymond, Robert C.

WELLINGTON

Diacon, James L.
McGovern, James L.

WICHITA

Bair, Albert E.
859 Fabrique Drive
Binter, Paul A.
2110 East Douglas
Blood, Mary J.
647 North Hillside
Brooks, H. W.
3306 East Central
Brown, Val J.
1550 North Madison
Browning, William H.
425 N. Hillside
Budetti, Joseph A.
1023 First National Bank Bldg.
Case, Laurel G.
845 Coolidge
Evans, Grant E.
435 North Hillside
Goodpasture, W. C.
2110 East Douglas Ave.
Grillot, Floyd B.
832 Woodlawn
Hagan, Daniel J.
1031 North Emporia
Hands, S. V.
618 South Oliver
Hiebert, A. E.
3306 East Central
Hoff, Donald E.
2721 Boulevard Plaza
Hull, Robert E.
5325 East Harry
McGuire, George E.
425 North Hillside

Morrison, G. B.
402 South Fountain
Nelson, Russell A.
1235 North Broadway
Owen, LaRue W.
608 Drury Lane
Padfield, R. E.
707 First National Bank Bldg.
Parker, C. A.
901 Nims
Petterson, Perry D.
2721 Blvd. Plaza
Pottorf, Mildred M.
114 North Oliver
Reals, William J.
3400 East Grand Ave.
Rossitto, A. F.
211 S. Hydraulic
Schweinfurth, James P.
3244 East Douglas
Scimeca, Salvatore A.
2021 North Market
Shrader, D. A.
706 Orpheum Bldg.
Smith, F. D.
1734 North Hillside
Swisher, William C.
133 North Glendale
Wood, Gary B.
2725 Boulevard Plaza

WINFIELD

White, R. Burnley

Out of State Physicians

ALABAMA

Haffner, V. B.
Montevallo

CALIFORNIA

Grider, W. H.
Altadena
Gyarfas, W. J.
Duarte
Floyd, Glen
La Jolla
Demand, J. W.
Long Beach
Ehrlich, Karl A.
Oakland
Peck, J. H. A., Jr.
San Diego
Tetzlaff, Fred M.
San Francisco
Helwig, G. F.
San Jose
Myers, Ben L.
Santa Rosa
Edwards, R. W.
West Los Angeles

COLORADO

Fall, N. B.
Colorado Springs
Marrs, E. A.
Colorado Springs

FLORIDA

Smith, Carl M.
Clermont
Coyle, John F.
Panama City

ILLINOIS

Vogt, William L.
Champaign

INDIANA

Taylor, James A.
Muncie
Ridlon, Albert M.
South Whitley
Hogan, T. W.
Terre Haute

IOWA

Aleshire, Irma
Iowa City

LOUISIANA

Howard, Richard R.
Slidell

MARYLAND

Pennington, Katherine
Baltimore
Bowen, Murray
Bethesda
Edwards, E. M.
Mt. Rainier

MICHIGAN

Hopp, Ralph L.
Ann Arbor

MINNESOTA

Selzer, Donald W.
Aitkin
Hildyard, V. H.
Minneapolis
Jastram, R. M.
Minneapolis
Maier, Edwin R.
Rochester

MISSISSIPPI

Ivy, H. B.
Meridian

MISSOURI

Martin, John N.
Chillicothe
Sterrett, Preston
Humansville
Garst, R. J.
Joplin
Weed, Randall
St. Joseph

KANSAS CITY, MISSOURI

Enna, Carl D.
Gundle, Sigmund
Johnson, L. D.
Mitchell, A. D.
Rumold, Mervin J.
Todd, Richard N.
True, Otis H.

MONTANA

Benkelman, Ward E.
Polson

NEBRASKA

Artman, John C.
Kearney

NEW MEXICO

Kridelbaugh, William W.
Albuquerque
Montgomery, Charles
Roswell

NEW YORK

Bishop, Francis E.
Massapequa
Coffey, Roy B.
New York City

NORTH DAKOTA

Weeks, Vernon L.
Fargo

OHIO

King, E. R.
Cleveland

OKLAHOMA

Harsha, W. N.
Oklahoma City
Stacey, Harley
Oklahoma City

PENNSYLVANIA

Claypool, J. Gordon
Narberth

SOUTH CAROLINA

Aronson, Gerald
Columbia

SOUTH DAKOTA

Peiper, Warren S.
Mitchell

TEXAS

Fairchild, John P.
Fort Sam Houston
Robison, Leonard J.
Irving
Nickell, Wendell K.
San Angelo
Wellman, Garland O.
Texas City

VIRGINIA

Crockett, C. A.
Arlington
Purinton, Lew W.
Norfolk

WASHINGTON

Treger, Donald M.
Bremerton
Wartman, Calvin
Bremerton
Voth, Harold W.
Larson AFB
Critchfield, T. W.
Seattle
Miller, Jack P.
Yakima

Deletions

Dorothy D. Allen, M.D. ..... Mankato, Kansas
Marian Barnes, M.D. ..... Osawatomie, Kansas
Harle Virgil Barrett, M.D. .. Leavenworth, Kansas
George Leo Basham, M.D. ..... Eureka, Kansas
Clarence Edwin Boudréau, M.D. El Dorado, Kansas
Archibald John Brier, M.D. ..... Horton, Kansas
Bernard Stephen Brody, M.D. .... Topeka, Kansas
William M. Brownell, M.D. .. Hot Springs, S. D.
William Hobart Burch, M.D. .. Ft. Bragg, N. C.
Robert Arthur Chapman, M.D. . Concordia, Kansas
Herbert L. Clark, M.D. ..... Topeka, Kansas
William Ebenezer Currie, M.D. .. Sterling, Kansas
Edward Idris Davies, M.D. La Canada, California
Donald Raymond Davis, M.D. .... Olathe, Kansas
Harry William Davis, M.D. ..... Plains, Kansas
Fred Erland Deal, M.D. ..... Weir, Kansas
Everette Lee Dye, M.D. ..... Manhattan, Kansas
Ellis Austin Evans, M.D. . Conway Springs, Kansas
Edgar Francis Ewen, M.D. ..... Chanute, Kansas
Edward L. Fitzgerald, M.D. .. Hutchinson, Kansas

Charles Mooney Fitzpatrick, M.D. .. Salina, Kansas
John W. Franklin, Jr., M.D. .... Chillicothe, Ohio
William Redmon Frisbey, M.D. .... Delia, Kansas
Jack Melvin Gilliland, M.D. .. Ft. Collins, Colorado
Robert Franklin Goodwin, M.D. .. Tacoma, Wash.
William C. Grasley, M.D. .... Charleston, W. V.
Edward Jacob Haerle, M.D. .. Minneapolis, Kansas
Thomas Newton Hall, M.D. .. Kansas City, Kansas
Lawrence John Hanis, M.D. .. Kansas City, Kansas
Robert Hawkins, M.D. ..... Marysville, Kansas
Schubert David Henry, M.D. . Kansas City, Missouri
Ralph Eugene Anthony Hines, M.D. Wichita, Kan.
Robert Bennett Hodgson, M.D. Prairie Village, Kan.
James Louis Hoopingarner, M.D. Cherryvale, Kan.
John L. Johnson, M.D. .. Birmingham, Alabama
John William Kelly, M.D. .... Louisburg, Kansas
Edward D. Kilbourn, M.D. .... Wichita, Kansas
Robert John Kinney, M.D. ..... Topeka, Kansas
Robert Miller Knox, M.D. ..... Des Moines, Iowa
Kenneth L. Knuth, M.D. .... Santa Ana, California
Albert Henry Krause, M.D. . Muskogee, Oklahoma
Abraham Martens Lohrentz, M.D. McPherson, Kan.
Carl Elijah Long, M.D. ..... Norton, Kansas
Orrin C. Lowe, M.D. ..... Paola, Kansas
David MacNaughton, M.D. Excelsior Springs, Mo.
James Thurston Makinson, M.D. LaCrosse, Kansas
Robert Lewis McAllister, M.D. Marysville, Kansas
Arthur Addison McAuley, M.D. Chateau, Montana
Marmaduke D. McComas, Sr., M.D. Courtland, Kan.
Elbert Daniel McNeil, M.D. ..... Satanta, Kansas
Richard Gordon Meisburger, M.D. Kansas City, Kan.
Charles Fredric Menninger, M.D. .. Topeka, Kansas
Clyde Otto Merideth, Jr., M.D. .. Emporia, Kansas
Carroll Dean Monroe, M.D. ..... Ulysses, Kansas
John Stewart Moore, M.D. .. Roswell, New Mexico
Dewey George Nemec, M.D. .... Agenda, Kansas
Malcolm C. Newman, M.D. . Yates Center, Kansas
William Brown Newton, M.D. Glendale, California
David Thomson Nicoll, M.D. Bronxville, New York
John Robert Patterson, M.D. Kansas City, Missouri
Thomas Rugely Perdue, M.D. . Bozeman, Montana
William Osee Poston, M.D. .... Quenemo, Kansas
Elmer L. Raynolds, M.D. ..... Mankato, Kansas
Jack William Revere, M.D. .. Grand Forks, N. D.
Murray E. Robinson, M.D. Klamath Falls, Oregon
George Edman Scheer, M.D. Staten Island, N. Y.
Bettye Sue C. Schurter, M.D. .. Hardtner, Kansas
Lonis Leon Schurter, M.D. ..... Hardtner, Kansas
John Robert Scott, M.D. .... Missoula, Montana
James Albert Settle, M.D. .... Hutchinson, Kansas
Joseph Shaffer, M.D. ..... Simpson, Kansas
Orlan Dayton Sharpe, M.D. .... Neodesha, Kansas
Charles Moses Siever, M.D. ..... Topeka, Kansas
Rheuben Christopher Smith, M.D. Marion, Kansas
William Kenneth Smith, M.D. .... Sedan, Kansas
Sherman O. Strand, M.D. ..... Wichita, Kansas
Joyce Randolph Sumner, M.D. Kansas City, Kansas
Adelbert Leon Suwalsky, M.D. Leavenworth, Kan.
Mowry Stafford Thacher, M.D. .... Turon, Kansas
John William Unruh, M.D. Prairie Village, Kansas
Leo Francis Wallace, M.D. Overland Park, Kansas
John Constantine Wilcox, M.D. Mulvane, Kansas
Lyle Ernest Wonderlich, M.D. Keesler, Mississippi
Orlin Pearl Wood, M.D. ..... Marysville, Kansas

Spelling Corrections

Attwood, George Arthur ..... Randolph
Glover, Harold Mortimer ..... Newton

Managing Editor Ill

Miss Pauline Farrell, managing editor of the JOURNAL, underwent surgery at St. Francis Hospital in Topeka on Wednesday, August 4. Her physicians report that she is improving rapidly, that she has left the hospital and is now convalescing at home. Her many friends in the medical profession, who will note the absence of her deft hand in the preparation of this JOURNAL issue, wish her a speedy recovery and an early return to her office.

Oklahoma City Clinical Society

The Oklahoma City Clinical Society which had its beginning in 1930 will open its twenty-fourth annual four-day Conference on October 25, 1954.

A large attendance is expected at this ever increasingly popular meeting held in centrally located and easily accessible Oklahoma City. It is interesting to note that at the present time Oklahoma City is rated third in the nation as a convention city.

As in former years, an outstanding program of postgraduate teaching has been arranged. This includes lectures and discussions by sixteen distinguished guest speakers selected from various medical and teaching centers throughout the nation, as well as many Oklahoma City teachers and physicians. Dr. Walter B. Martin of Norfolk, Virginia, president of the American Medical Association, will give an address at the first banquet meeting October 25th. In addition to the general assemblies and panel discussions there will be daily luncheon round table question and answer sessions, and a clinical pathologic conference. The entertainment will include dinner meetings, the annual Clinic Dinner Dance and the Stag Smoker.

A cordial invitation is extended to all physicians who are members of their County Societies to attend this meeting from October 25 through October 28.

Clinicopathological Conference

Case Presentation

Dr. Mahlon Delp, professor of medicine: Dr. Harry S. N. Greene, professor of pathology at Yale, is the visiting professor of pathology at our institution presently and is to be our guest pathologist today. I would hope that our clinicians will be circumspect of their time so that we won't infringe upon the pathologist's time as we have done sometimes in the past. Dr. Steinzieg is going to give us the history.

Dr. Sherman Steinzieg, resident in medicine: The case today is that of a 72-year-old white male who was admitted to KUMC for the first time on June 11, 1953, and who expired on June 14, 1953.

The chief complaint was coma on admission.

Regarding the past history, the patient's family stated that he was apparently in good health until approximately one year prior to admission at which time he developed intermittent bouts of epigastric pain. He also complained at times of a lump in his abdomen. Two months prior to admission the patient became anorexic and developed more pronounced abdominal cramps. He was admitted to another hospital where he rapidly became jaundiced and subsequently developed severe anorexia and dysphagia. He lapsed into coma just prior to being admitted to this hospital. The patient experienced an illness characterized by transient jaundice about five years prior to death. Six months prior to death he underwent prostatic surgery because of difficulty in urination.

Physical examination showed the blood pressure to be 130/75, pulse 80 and regular, temperature 99.6 orally. Pupils reacted sluggishly to light. Bilateral cataracts were present. Fundi were not visualized. The sclerae were icteric. The mouth was edentulous; the tongue was dry; ears, nose and throat were negative. The thyroid was not palpable. There were no unusual cervical masses.

Harsh bronchial breathing was noted in the right posterior lung field and a questionable friction rub was heard. Breath sounds were generally diminished. The lung fields were otherwise clear to percussion and auscultation. The heart was not enlarged to percussion. A_2 was greater than P_2. No murmurs, thrills or arrythmias were noted.

The liver edge was firm and easily palpable three centimeters below the right costal margin. A soft 5 by 5 centimeter mass was noted in the right upper quadrant of the abdomen. The spleen was not palpable.

The extremities were quite emaciated, and the fingers and toes were cyanotic. Deep tendon reflexes could not be elicited. Rectal examination disclosed a slightly enlarged, firm, nodular prostate.

Laboratory examination on June 11 showed that the urine was acid in reaction, specific gravity 1.009, albumin 2 plus, sugar negative, microscopic fields loaded with granular, hyaline and pus casts with 8-10 pus cells per high power field. Blood count was 4.35 million with 12.2 grams hemoglobin; white blood count of 17,150 with 88 per cent polymorphonuclear leucocytes (2 non-filamented), 10 per cent lymphocytes and 2 monocytes. VDRL was negative. Sedimentation rate was 27 millimeters in one hour. NPN 175 mg. per 100 cc., creatine 5.6 mg. per 100 cc., blood sugar 158 mg. per 100 cc. (blood drawn while IV glucose was administered), serum sodium 147 mEq/L., potassium 3.5 mEq/L., CO_2 13.5 mEq./L., chloride 132 mEq/L. Acid phosphatase 1.2. Serum bilirubin, direct, 49.2 mg. per 100 cc., total 70.7 mg. per 100 cc., alkaline phosphatase 54.2, urinary urobilinogen 0.8 milligrams (280 cc. total volume), cephalin cholesterol 4 plus, thymol turbidity 16 units, serum albumin 3.5, serum globulin 4.0, total cholesterol 932 with 10 per cent esters, prothrombin time 25 per cent of normal. On June 12, 1953: NPN 195, creatine 6.8, fasting blood sugar 132, sodium 160, potassium 4.1, CO_2 11.9, chlorides 135.

On admission it was obvious the patient was terminal. However, he was given vigorous supportive therapy, which included blood transfusions, intravenous fluids, antibiotics (penicillin and streptomycin) and 200 milligrams of cortisone intramuscularly. On the night of admission, the patient's temperature per rectum rose to 105.8. He developed a hypotension which was treated with blood transfusions and additional fluids. The following morning the rectal temperature was down to 99.6. However, the patient's condition deteriorated.

On the second hospital day a Levine tube was placed in the stomach for feeding purposes. On the evening of the second hospital day, the rectal temperature again rose to 103.8 and the blood pressure fell to shock levels. In spite of treatment with antibiotics, fluids, blood transfusions and Levophed (nor-epinephrine), the patient's condition worsened

From the University of Kansas Medical Center, Edited by Glen R. Shepherd, M.D., and Mahlon Delp, M.D., from recordings of the conference participated in by the departments of medicine, radiology, surgery (urology) and pathology, and the third and fourth year classes of medical students.

with a quiet death occurring at 8:30 a. m. on June 14, 1953, the third hospital day.

Dr. Delp: Are there questions now concerning the protocol?

Dr. Max Allen, associate professor of medicine: On the second morning, was any difficulty experienced in passing the Levine tube?

Dr. Steinzieg: There was no difficulty about the Levine tube.

Q: The 280 cc. total volume for the urobilinogen determination was representative of a 24 hour output?

Dr. Steinzieg: No, his urinary output was fairly good. It averaged between 1000 and 1500 cc. daily during the three days that he was here.

Q: Was the coma quiet or disturbed?

Dr. Steinzieg: Quiet.

Q: What kind of bowel movements did he have?

Dr. Delp: We have little information concerning the bowel movements prior to his hospitalization. He was having tarry stools the last few hours before his death.

Q: He didn't have tarry stools prior to his hospitalization?

Dr. Delp: We don't think he did have.

Q: Were any spider nevi or distended veins noted over the abdomen?

Dr. Delp: No.

Q: Was there an amine odor of the breath at any time.

Dr. Delp: Yes, he did have an amine odor of the breath from the time of entrance.

Q: Was there any other evidence of hemorrhage?

Dr. Delp: No. This patient was running a fever, he had a temperature of 105.6 and 105.8 within a matter of two or three hours after he arrived here. His temperature curve is pretty typical of what you see on the sheet, sustained but with high peaks. Any other questions? All right, thank you Dr. Steinzieg.

We have one EKG. Stevens, will you demonstrate it for us?

Phillip Stevens, fourth year student:* I was not given the date of this EKG. It shows a rate of approximately 80 with regular sinus rhythm. The most striking features of this electrocardiogram are the slight depressions of ST segments in leads 2, 3, and AVF with a flattening of the T waves in almost all the leads and with prolongation of the QT interval. The diagnosis on the basis of these three findings is hypopotassemia. We cannot rule out a prolonged QT interval on the basis of a hypocalcemia.

Dr. Delp: Any further comments about this, Dr. Dimond.

Dr. E. Grey Dimond, professor of medicine: No.

Dr. Delp: Wilhelm, do you have the x-rays?

* Received M.D. degree in June, 1954.

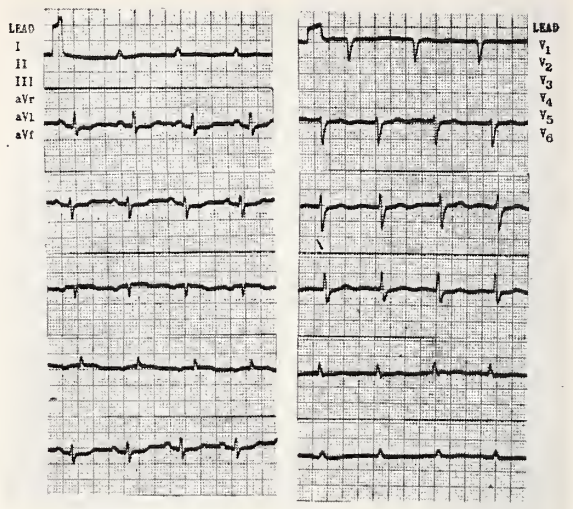


Figure 1. EKG, showing normal sinus rhythm with depressions of ST segment, T wave flattening, and QT prolongation suggesting hypopotassemia.

Paul Wilhelm, fourth year student:* These were taken on the day of admission. The chest film shows that the heart is normal. The costovertebral angles are clear. I can see no pathology of the bony structure. The aorta is prominent which one might expect in a man this age. The bronchial markings are very clear. No pulmonary infiltration is seen.

On the KUB film, the most marked thing is a scoliosis of the lumbar spine and osteoarthritic changes.

* Received M.D. degree in June, 1954.

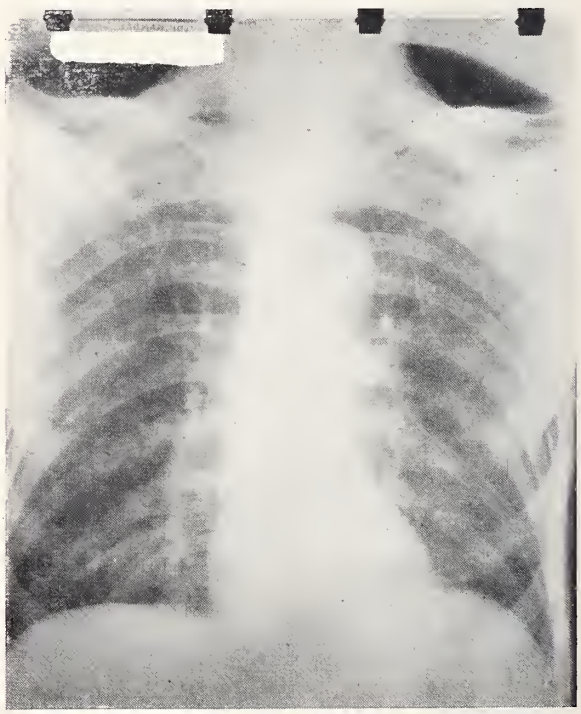


Figure 2. X-ray of chest showing degenerative changes.

Dr. Delp: Dr. Hogan, do you have any comments on the x-ray films?

Dr. Thomas Hogan, resident in radiology: I think the films have poor technique and the detail is somewhat decreased because of that. We interpreted them as essentially normal except for degenerative changes in the spine.

Dr. Delp: You didn't actually think that there was any infiltration in the lung?

Dr. Hogan: No, it wasn't so interpreted.

Dr. Delp: Now, for the purposes of the differential diagnosis and the clinical discussion, I would like to recall certain items in the protocol. First of all, this was a 72-year-old individual. He had been comatose for 24 hours prior to his arrival here. In the history, aside from the coma which his family reported and which we easily observed, he had a loss of appetite and this was emphasized. He had had abdominal pain and jaundice for two months. Whether or not it is significant, I mention that this patient had an episode of jaundice five years prior to this illness. Surgery had been done on his prostate, I assume a transurethral resection, six months prior to this KUMC hospital admission which would make it four months prior to the onset of the present illness.

I saw this man within a matter of minutes after he came in. These features of the physical examination I think were quite obvious. He was jaundiced, comatose, with a mass in the abdomen and certain chest findings which have been recorded in the protocol. There were a number of rather marked elevations and alterations in laboratory findings including the NPN and other features of the liver function profile. Their significance, I think depends upon your own interpretation. Actually I suppose the clinicians are obligated to engage in a rather old academic exercise to determine whether this is an obstructive type of jaundice, a surgical jaundice, and/or a medical type of jaundice. It would seem that there are several possibilities. Lastly of all, I think we are again obligated to attempt to explain the patient's death at the time that it actually occurred. Weigel, will you begin our discussion?

DIFFERENTIAL DIAGNOSIS

John Weigel, fourth year student:* We must do three things: consider causes of coma, causes of this patient's illness and the causes of his death. As



Figure 3. Photograph of gross specimen of tumor, showing opened distended common bile duct with constricting mucosal lesion just above ampulla. A block has been taken from the area of the tumor.

* Received M.D. degree in June, 1954.

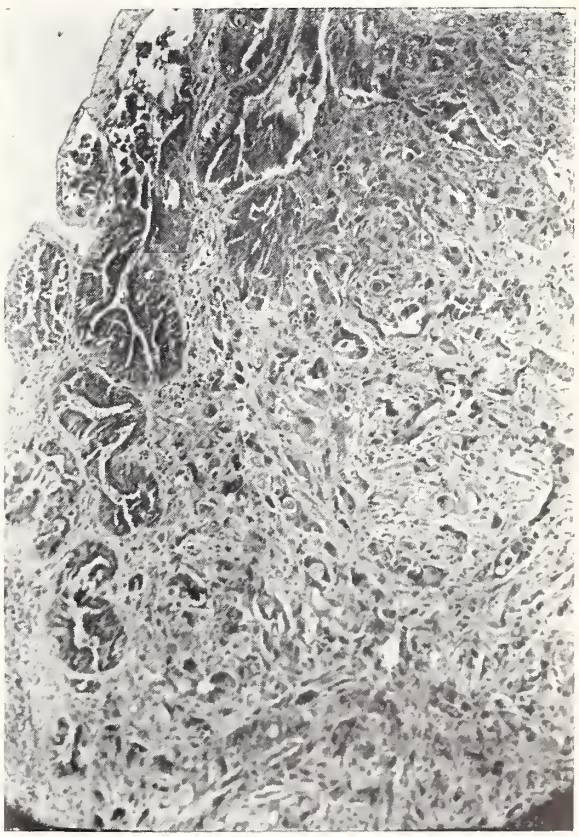


Figure 4. Photomicrograph of common bile duct showing infiltration of wall by tumor and its acinar structure.

Dr. Delp has briefly reviewed his symptoms, I won't repeat them.

As to causes of coma, I think we can run very quickly through a good number of these. CVA's and cerebral neoplasms I think we can rule out on lack of history and on the slow onset. Subdural hematomas and middle meningeal artery hemorrhage we can rule out on the history, on the slow onset, and on the physical findings of jaundice in this man's history. This man presented no history of having taken drugs and this was a slow onset. It lasted for a number of days.

This leaves us with a metabolic cause of coma which are several in number. First of all, diabetic coma. Again we have no history of diabetes and we don't have an elevated blood sugar.

We are left with two things, hepatic coma and renal coma. I think I'll confine my discussion pretty much to those two. First of all I'd like to talk a little bit about hepatic coma. This usually begins with a period of excitement or lethargy leading into coma and it's often fairly insidious in onset. This patient had jaundice and fetor hepaticus or amine breath which is again suggestive of liver damage. Oftentimes all other organs of the body suffer.

These patients can present with shock and collapse of the cardiovascular system. This patient had shock. They can present with urinary tract findings such as anuria or oliguria. Probably it is on the basis of shock and failure of the kidneys to do their work. Such patients also present as just a generalized metabolic acidosis which is usually uncompensated. This man had deep respirations which again goes along with the acidotic condition and dry mucous membranes.

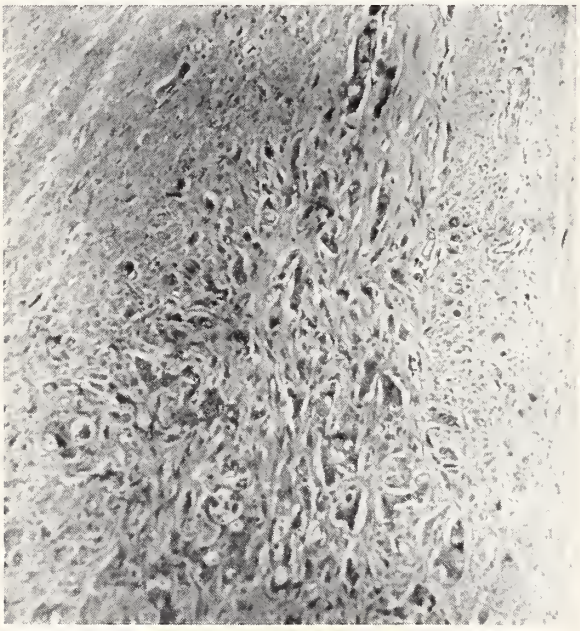


Figure 5. Photomicrograph showing higher power view of anaplastic area of the carcinoma.

There have been several mechanisms postulated concerning liver failure. I think one of them is failure of the liver cells to phosphorylate thiamine. Thiamine, of course, is necessary in the tricarboxylic acid cycle and if it doesn't work the patient cannot metabolize the glucose properly. This generally comes out in some of the laboratory work which wasn't done on this patient. Usually there is an increase in lactic acid and pyruvic acid. The urea, the high NPN, again is compatible with renal failure on the basis of hepatic coma. Another mechanism postulated in hepatic coma is intracellular ammonium ion poisoning. It has been shown that a number of patients in hepatic coma will have increased serum ammonia. It is difficult to tell what their intracellular ammonia is but it's known that intracellular ammonia acts as a neurotoxin.

As to the cause of possible hepatic coma in this man, I think we have to think of three things: prehepatic disease, hepatic, or posthepatic disease. I assume on the basis of very high cholesterol and on the basis of the high alkaline phosphatase, high bilirubin and the fact that he had a palpable mass here—which I interpreted as his gallbladder—that he had biliary tract obstruction, probably posthepatic. I think that in cases where you can feel a large gallbladder the odds are that something is blocking the common duct and probably the obstruction is there. Some of these liver tests do show severe hepatocellular damage such as the flocculation test. The alkaline phosphatase elevation could result from intrahepatic blockage. So I think all of these liver tests show us two things: one is that there is obstructive jaundice and the other is that there is a high degree of hepatocellular damage. The urinary urobilinogen finding, which in this man is in the upper limits of normal, is rather confusing but I think that somehow a little bile must have leaked through.

As to the causes of posthepatic biliary obstruction, I want to just review the fairly prominent ones. First of all we have to think of stones as most common. This man did have an episode of transient jaundice five years ago but that's all we have. We don't have an x-ray finding of stones but that doesn't rule it out. But the type of pain, the general lethargy, and the anorexia are not the type of symptoms commonly associated with gallbladder colic. It is also more common in women. This predilection for women applies also to carcinoma of the biliary tract, carcinoma of the gallbladder. That usually occurs in women. It is found 80 per cent or more of the time with gallstones. You wouldn't expect a large, soft gallbladder in that disease.

As far as carcinoma of the pancreas is concerned, we have to think of carcinoma of the head of the pancreas because that's the common cause of the onset

of jaundice in such a patient as this, a man with pain. I think it could be present but there are certain things that would tend to rule it out. I think this patient's other complaints would tend to rule out a primary carcinoma at the head of the pancreas. As far as the tail and body of the pancreas is concerned we'd expect possible diabetes with destruction of the pancreatic gland.

Carcinoma of the ampulla of Vater occurs and again we have a rather insidious onset of generally painless jaundice and also we have bleeding of the GI tract. This man does have bleeding of the GI tract but he did not have a painless jaundice. Metastatic disease to the liver—and I'll talk about this a little more later—is a very possible cause of extrinsic pressure on the common duct which could cause biliary obstruction. Cholangitis commonly follows stasis from any cause. I think the high fever and evidence of liver damage suggests a suppurative cholangitis with extensive destruction of liver tissue.

Renal coma deserves consideration. Its symptoms occur gradually and include lethargy. These patients present with vomiting, anorexia, and apathy. They develop acidosis often with a loss of calcium. They may present as having convulsions. Occasionally they will lose potassium and that could account for this patient's lowered potassium. They can lose potassium by vomiting, by diarrhea as well as by renal mechanisms.

Chloride acidosis can exist in terminal renal disease simply by giving these patients too much chloride, the kidney being unable to handle it. This could account for this man's increased chloride. He did have increased chloride and he had had IV's given. I think the great increase in the NPN, the positive albumin, the cells and the pus casts are all indicative of renal disease. Particularly important, I think, is the creatine being as high as it is. Specific gravity appears to be rather fixed also. The NPN could be due partly to resorbed blood from the GI tract. A prerenal azotemia could be present on that basis but I don't think that would account for the tremendous elevation of NPN which coupled with the other renal findings makes us think of a severe renal disease.

This man had no anuria but he did have casts which make you think of tubular damage, though not lower nephron nephrosis. Cholemic nephrosis is a condition that oftentimes occurs in regurgitation or posthepatic jaundice in which a good deal of bilirubin and bile products are in the blood. It is postulated that bile salts are toxic to the tubules and cause tubular breakdown. There again we see casts, pus cells, epithelial cells.

Hepatorenal syndrome has been discussed by a number of men. Whether such a thing exists or not I don't know. Possibly the shock that occurs in liver failure could account for anuria, oliguria and the signs of renal shutdown as well as the toxic products.

The most common cause of kidney disease in a man over 50 with history of prostatism and prostatic surgery as this man had is pyelonephritis on the basis of obstruction. Obstructive pyelonephritis is roughly 12 times as common as blood-born pyelonephritis. I think he probably developed mild or moderate hydronephrosis bilaterally. I think he probably developed ascending urinary infection with pyelonephritis in both kidneys. This man could have had hypertension on a renal basis and have the symptoms masked by the fact that he was in shock part of the time and on the borderline of shock.

With the history of a nodular prostate and acid phosphatase of 1.2, we just about have to say that this patient had carcinoma of the prostate. I think that that could have metastasized to the liver very easily and caused the obstruction there. In reported cases of carcinoma of the prostate, a good number have metastasis to the lungs and liver.

So my final diagnosis is primary adenocarcinoma of the prostate gland with metastases to the liver causing biliary obstruction. He probably had an ascending suppurative cholangitis destroying a good deal of his liver. I think he had bilateral pyelonephritis with destruction of kidney parenchyma. I think he died of metabolic breakdown or ammonia intoxication.

Dr. Delp: Dr. Mitchell what comments can you make about the acid phosphatase values which are so disturbing?

Dr. Andrew Mitchell, assistant clinical professor of surgery (urology): The acid phosphatase, I presume, is reported in millimoles, which means that it's double the normal value and would be considered pathognomonic of carcinoma of the prostate. I think the occurrence of carcinoma of the prostate in a man 72 years old would be frequent enough that the likelihood of his having two diseases would be greater than the likelihood of the carcinoma of the prostate explaining all the present picture. I would say that his renal manifestations were on a basis of preexisting renal damage which probably antedated his resection. He had gone beyond a point where he would recover simply from having the obstruction removed. He probably had two diseases. This terminus for carcinoma of the prostate would be much rarer than the coincidental finding of carcinoma of the prostate. Probably from 20 to 30 per cent of all 72-year-olds have carcinoma of the prostate.

Dr. Delp: You wouldn't be disturbed by the absence of any bony lesions appearing in the x-ray films?

Dr. Mitchell: No, I wouldn't because he could have had enough lymphatic metastases to explain that elevation of acid phosphatase. He wouldn't have had to have bony lesions. Of course that's not much

of a KUB x-ray to rule bony lesions in or out on, but I don't think the whole thing can be explained on that basis. As far as his shock was concerned, one morbid finding of the carcinoma of the prostate and one not too greatly publicized is involvement of the adrenals. A good many people terminate their carcinoma of the prostate from adrenal failure with replacement by carcinoma of the prostate.

Dr. Delp: Now I'm just a little disturbed about the trend the clinical discussion has taken here. Dr. Rankin, I want to ask you your opinion concerning the possibility of intrahepatic metastatic lesions producing a blood cholesterol of 900 and an alkaline phosphatase of 50.

Dr. Thomas J. Rankin, assistant professor of medicine: As far as experience is concerned in the literature, it is very uncommon for alkaline phosphatase and cholesterol, both of which are phenomena seen in biliary obstruction, to rise to these levels from intrahepatic disease. The question has been asked heretofore by Dr. Delp if this might be a homologous serum jaundice. In common with all the rest of us I cannot in any terminal disease of the liver of this degree make that differential diagnosis. I am much inclined to feel that the thing is extrahepatic biliary obstruction because of the extreme level of the total cholesterol, because of the extreme level of the jaundice, and because of the extreme level of the alkaline phosphatase. As far as the clinical picture is concerned, I would certainly feel it to be clinically rather typical of so-called hepatorenal syndrome.

I cannot, of course, state how much of renal disease was due to chronic pyelonephritis which a man of this age with benign prostatic hypertrophy must have, but I think that the rapidly rising NPN and the terminal picture do certainly suggest the so-called hepatorenal syndrome. Since the man passed from 1000 to 1500 cc. of urine, I think that acute renal failure is less likely than the so-called bile nephrosis.

I believe then he had two causes for his renal failure: the original chronic pyelonephritis and the superimposed renal damage which accompanied his hepatic disease. I think time is too short to discuss the causes of death in hepatic coma and in azotemia. They are very confused and nobody can select the actual metabolic reason. I do certainly think that they do play primarily upon the central nervous system and the basal ganglia as one of the mechanisms of death.

Dr. Delp: May we hear from the pathologist, Dr. Joffe.

PATHOLOGY REPORT

Dr. Irwin Joffe, resident in pathology: There was a tumor which diffusely infiltrated the head of the pancreas and also involved the terminal one and one-half centimeters of the common bile duct. The tumor also involved the mucosa of the duodenum in the vicinity of the ampulla of Vater, involving just a small area about one centimeter square. The lesion in the head of the pancreas was poorly delimited and was a greenish-yellow in color. It appeared to involve grossly the root of the mesentery. The tumor involving the distal end of the common bile duct, which incidentally was not completely occluded, involved the mucosa and submucosa. In the area examined, it appeared not to penetrate the muscularis to involve deeper structures but some of this material, as you will see in the specimen, remains for your examination. It seemed likely to me that in some areas there was connection between the lesion in the distal common bile duct and the head of the pancreas. There was dilatation of the pancreatic duct, the duct of Wirsung, to a maximum circumference of three centimeters. The common bile duct was greatly dilated above the constricted lower portion to five centimeters in diameter. The cystic duct was two centimeters in greater circumference and the gallbladder was greatly enlarged, measuring 15 x 7 cm.

There were small masses of apparently thrombotic material partly occluding the superior mesenteric vein and the inferior vena cava in the vicinity of the pancreas. Another thrombotic mass was found in the right branch of the portal vein. Associated with this there were incomplete focal infarcts in the right lobe of the liver.

There was, of course, jaundice of all organs. The kidneys particularly were jaundiced and somewhat swollen. They weighed 420 grams together. The cortical medullar junctions were somewhat indistinct and the calyces on the left were slightly battered. The ureters were not dilated, the bladder was quite small and showed a mild acute cystitis. The prostate was small. It showed no evidence of recent operation and no evidence of nodular hyperplasia of any significance and no carcinoma grossly.

Dr. Harry S. N. Greene, professor of pathology at Yale: Several years ago following statements that autopsy was a much overrated pastime, my staff suggested that I was a disturbing influence at clinicopathological conferences and it might be well if I stayed away. This sort of thing is getting to be an unusual experience with me.

Morphologically in this case, interest centers around a tumor in the head of the pancreas. The question of interest from the pathological or academic point of view is the location of the point of origin. Is it in the head of the pancreas, pancreatic duct or in the common bile duct? I think that usually can be fairly well guessed at from the morphology.

There are two types of tumor structure. One area shows a rather anaplastic tumor. Quite obviously it is an adenocarcinoma but quite disorganized and undifferentiated.

Other areas show a good deal more organization

and definite acinar-like structures, an adenocarcinoma without question. Well, that doesn't help any, that may come from any one of those places. The head of the pancreas itself was a seat of a widespread chronic fibrosis, with not much indication of an origin. However, it could be the head of the pancreas. The pancreatic ducts were very interesting.

Frequently one found inside the ducts areas of polypoid hyperplasia which superficially and under low power looked something like cancer. However, they're not cancer. These were well differentiated cells and it was simply the hyperplasia appearing in those ducts following obstruction.

The epithelial surface was absent but in its wall were found structures which duplicated very well the acinar structures found in the tumor. However, whether this was invasion of the wall of the common bile duct from outside or from epithelia that desquamated simply can't be told. Thus, we're left in the dark as to the origin of this tumor, whether it was from the head of the pancreas or from the common bile duct.

A very interesting feature of this tumor was its widespread extension along lymphatics, particularly along perineural lymphatics.

There were thrombi in various regions in the inferior vena cava and periportal veins and inside of these thrombi there were growing cancer cells and also cancer cells in the walls of the vein. There were one or two minute lesions in the liver which could be construed as metastases. There were, however, no metastases elsewhere in the body.

The liver showed, as might be expected, evidences of an obstructive jaundice, some fibrosis and bile products.

Cholemic nephrosis of the kidney, degeneration of the epithelium and proximal tubules, and bile stained casts appeared in those slides.

There were masses of growing bacteria in the pneumonia of the lung.

So, from a biological point of view, interest in this case is in the fact that there were so few metastases. There was widespread extension of the tumor in lymphatics but metastasis was limited to two or three questionable lesions in the liver.

There is a tremendous difference between lymphatic extension and metastasis. Every time a cell undergoes mitosis the pressure inside the mass increases. The growing cells therefore take the path of least resistance in their continued extension. If there happens to be an open tissue space or a lymphatic, it will proceed to the regional lymph node where it will expand into a large mass. Tumors can extend for tremendous distances along lymphatics. I recall a case, for example, that showed extension of a breast carcinoma all the way down to a popliteal lymph node by means of lymphatic extension.

Metastasis is an entirely different circumstance. It's a discontinuous growth. It requires the tumor's having undergone a much longer development. It not only is capable of extension, it is capable of true invasion. There is a difference between extension and invasion.

One case of mine showed extension of tumor along tissue spaces between muscle fibers. If the muscle fiber is caught between two expanding masses it undergoes pressure atrophy and the tumor grows into that area. That is simple tissue occupation, the way a basal cell tumor grows.

True invasion is somewhat different and it's important to distinguish between invasion and extension for, as far as I am aware, the appearance of true cellular invasion is the only morphological criterion of metastasizing ability in the tumor. Unfortunately it can't be found in all situations for in order to find it, the cells adjacent to the tumor must be bigger than the tumor cells themselves.

Invasion is one of the necessities of metastasis. The tumor cell, in order to metastasize, must possess the ability to invade vascular walls. Once it gets inside a blood vessel it must possess the ability to survive living in the normal blood stream of the individual. In addition, after it has lodged in some endothelial surface, it must have the ability to invoke a stroma locally and then to grow extravascularly.

Well, how about this case, what was missing? Was the tumor autonomous? We don't know because no test for autonomy was made. Tumors of the common bile duct do become autonomous and do metastasize.

We know that tumor cells were present in the blood stream because we found them in growing thrombi. Why then, weren't there more metastases? Perhaps it's concerned with the inability of the tumor cells that were circulating in the blood stream of this individual to obtain a stroma. And stromatization of circulating tumor cells is extraordinarily important in the formation of metastases. Stromatization of circulating tumor cells is determined by exactly the same factors that govern stromatization of the graft.

Stromatization of tumor or graft cells depends on two factors: first, the ability of the cells to invoke a stroma, and second, the responsiveness of the connective tissue at the transplantation site. Both of those factors vary a great deal.

The ability to invoke stroma varies enormously. Some parenchymatous cells, for example those of the kidney or of the breast, are good stroma inducers. Other cells, such as those of the adrenal or the brain, are tremendously poor stroma inducers. As a matter of fact, ability to induce stroma seems to be directly proportional to the amount of connective tissue required by the parenchymous cells to form an adult structure. The kidney and the breast are complicated

admixtures of stroma and parenchyma and their cells are good stroma inducers. On the contrary the brain and the adrenal have a very small stromal content and in turn are poor stroma inducers.

The ability of connective tissues at various sites also differs widely in its ability to respond to a stroma inducing stimulant. If, for example, one takes a poor stroma inducer such as the embryonic adrenal and puts it into the subcutaneous space of an animal, it obtains no stroma whatsoever. The poor stroma inducing capacity of that adrenal is not sufficient to invoke a stroma from the very insensitive connective tissue of a subcutaneous space. Perhaps the most insensitive connective tissue in the whole body is that of the subcutaneous space and that is one of the reasons why skin transplantation is so difficult. However, if the embryonic adrenal is transferred to the anterior chamber of the eye, the poor stroma inducing stimuli are sufficient to invoke a stroma from this very responsive connective tissue in the iris.

Occasionally one finds tumors that have extraordinarily poor stroma inducing capacities. An outstanding example is the hypernephroma of the kidney. Such a kidney may grow to enormous size in an individual, actually become autonomous, invade the renal vein and at the time of operation it's obvious that there must be tumor cells in the blood stream. Yet after operation, such a patient may do perfectly well and never show metastases. And that is due to the fact that the cells of a hypernephroma are such poor stroma inducers.

If one transplants, for example, an autonomous hypernephroma from a human being into a guinea pig's eye, the cells grow but they grow in a manner of tissue culture, imbibing nutrient from the surrounding aqueous tumor. However, if such as tumor is transplanted to the brain, the extraordinarily responsive connective tissue of the brain will respond to this stimulus and a stroma will form.

The situation of the brain is extremely interesting in passing. Here one has a parenchyma which is probably the poorest stroma inducer there is, but a connective tissue which is the most responsive. If the situation were different and if in the brain we had a parenchyma that was a good stroma inducer, we'd undoubtedly have many more fibrous brains and a great deal more trouble getting along with clinicians. As a matter of fact, that is the reason why the brain tumors, the glioblastoma multiforme and the medulloblastoma, never metastasize. The tumor cells do get into the blood stream but they are such poor stroma inducers that they will not induce a stroma in any connective tissue in the body other than that of the iris and the brain.

Let me mention how some of these tumors can be transplanted, a tumor of a very poor stroma inducing capacity such as the hypernephroma. We can transplant them by means of a middle man and this technique illustrates very well, I think, the two factors concerned in transplantation and metastases: stroma inducing abilities and connective tissue responsiveness. We've been able to transplant such poor stroma inducers as hypernephroma to the anterior chamber of the eye by using the middle man. Embryonic tissue contains a very responsive connective tissue and it's also a good stroma inducer. The technique therefore is then a double transplantation. We first transplant embryonic guinea pig lung to the anterior chamber of the eye. That tissue, being a good stroma inducer, induces stroma in the adult iris. Then later on, we transplant the hypernephroma to the embryonic transplant. The embryonic connective tissue in the lung, being such a good and responsive connective tissue, responds to this poor stroma inducing stimulus and gives it a stroma.

The question remains why there were so few metastases in this individual? It would appear that the tumor was autonomous. It got into the blood stream and undoubtedly survived circulation in the blood stream. Now, not all autonomous tumors have that capacity. Not infrequently an animal tumor, one that is metastasized in the susceptible host, if removed and ground into emulsion and injected into the blood stream of a normal animal produces nothing whatsoever. The tumor cells are killed by the normal blood stream. It has been presupposed in many cases that some alteration must exist in the blood stream of a patient with metastatic tumor to allow metastasis to occur.

The other possibility in this case is that the tumor cells circulated but did not become stromatized. This is an adenocarcinoma and should be a good stroma inducer. But perhaps something happened to the connective tissue in this individual which made it unresponsive. I've no answer to this whatsoever but it is a fact, I believe, that in patients with arteriosclerosis, metastases are comparatively rare. They do occur but never in the abundance that they do in normal individuals.

ANATOMICAL DIAGNOSIS

Primary

Adenocarcinoma of the common bile duct with extension into the root of the mesentery, the descending portion of the duodenum, the head of the pancreas, the liver, celiac and peripancreatic lymph nodes and sympathetic nerves and ganglia.

Dilatation and polypoid hyperplasia of the duct of Wirsung and dilatation of the major extra and intrahepatic bile ducts, advanced with bile stasis.

Atrophy of parenchyma of the pancreas, advanced (history of fasting blood sugar 132 mg. per 100 cc.).

Organizing thrombosis of the portal veins and inferior vena cava containing proliferating tumor cells

Dramamine's® Effect in Vertigo

Dramamine has become accepted in the control of a variety of clinical conditions characterized by vertigo and is recognized as a standard for the management of motion sickness.

Vertigo, according to Swartout, is primarily due* to a disturbance of those organs of the body that are responsible for body balance. When the posture of the head is changed, the gelatinous substance in the semi-circular canals begins to flow. This flow initiates neural impulses which are transmitted to the vestibular nuclei. From this point impulses are sent to different parts of the body to cause the symptom complex of vertigo.

Some impulses reach the eye muscles and cause nystagmus; some reach the cerebellum and skeletal muscles and righting of the head results; others activate the emetic center to result in nausea, while still others reach the cerebrum making the person aware of his disturbed equilibrium. *Vertigo may be caused by a disease or abnormal stimuli of any of these tissues involved in the transmission of the vertigo impulse, including the cerebellum and the end organs.*

A possible explanation of Dramamine's action is that it depresses the overstimulated labyrinthine structure of the inner ear. Depression, therefore, takes place at the point at which these impulses, causing vertigo, nausea and similar disturbances, originate. Some investigators have suggested that Dramamine may have an additional sedative effect on the central nervous system.

Repeated clinical studies have established Dramamine as valuable in the control of the symptoms of Ménière's syndrome, the nausea and vomiting of pregnancy, radiation sickness, hypertension vertigo, the vertigo of fenestration procedures, labyrinthitis and vestibular dysfunction associated with antibiotic therapy, as well as in motion sickness.

Any of these conditions in which Dramamine is effective may be classed as "disease or abnormal stimuli"* of the tissues including the end organs (gastrointestinal tract, eyes) and their nerve pathways to the labyrinth.

Dramamine (brand of dimenhydrinate) is supplied in tablets of 50 mg. and liquid (12.5 mg. in each 4 cc.). It is accepted by the Council on Pharmacy and Chemistry of the American Medical Association. G. D. Searle & Co., Research in the Service of Medicine.



The site of Dramamine's action is probably in the labyrinthine structure.

*Swartout, R., III, and Gunther, K.: "Dizziness:" Vertigo and Syncope, GP *8*:35 (Nov.) 1953.

and organizing thrombosis of the superior mesenteric vein.

Focal incomplete infarcts of the right lobe of the liver.

Pericholangiohepatitis, slight.

Generalized jaundice, advanced.

Cholemic nephrosis, advanced.

Focal hemorrhages in the gums, submucosa of the small and large intestine, adventitia of the root of the aorta, beneath the pleurae of the lower lobes of both lungs, and in the skin (history of prothrombin time of 25 per cent of normal).

Emaciation.

Acute focal esophagitis (history of gastric intubation, 1 day).

Traces of gastric contents and mucus in trachea (history of coma, 3 days).

Bronchopneumonia of all lobes of both lungs, advanced, weight 2570 grams, with aspiration bronchopneumonia in upper lobe of left lung.

Dilatation of the heart, slight.

Acute passive congestion of the liver and spleen, moderate.

Ascites, 100 ml.

Acute cystitis, slight (history of indwelling catheter, 3 days).

Accessory

Arteriosclerosis of the left anterior descending coronary, basilar and left vertebral arteries, advanced; of the left circumflex and right coronary arteries, aorta, both middle cerebral and right vertebral arteries, moderate; of the anterior cerebral arteries, slight.

Sclerosis of the anterior leaflet of the mitral valve, slight.

Healed intramural anteroseptal myocardial infarct, small.

Interadherence of the cusps of the aortic valve, slight.

Fibrous thickening of the appositional surfaces of the mitral valve, slight.

Calcified lymph node at the hilum of the right lung.

Fibrous pleural scar of the apex of the upper lobe of the right lung.

Fibrous pleural adhesion between upper and lower lobes of the left lung.

Emphysema of the anterior borders of both lungs, moderate.

Arterial and arteriolar nephrosclerosis, moderate.

Chronic pyelonephritis, left, slight.

Retention cysts of the left kidney, slight.

Multiple diverticula of the colon.

Acute ulcerative proctitis, moderate.

Pedunculated polyp of the sigmoid colon.

Adrenal rest in region of celiac ganglion.

Accessory spleen.

Partially edentulous mouth.

Arcus senilis.

ACTIVITIES OF MEMBERS

Dr. and Mrs. D. V. Conwell, Wichita, are touring the continent of Europe following Doctor Conwell's presentation of a paper at a medical meeting in London, England.

Upon completion of a surgical residency in Research Hospital, Kansas City, Missouri, Dr. James D. Colt V, has resumed his association with his father, Dr. J. D. Colt, Jr., in Manhattan.

Dr. Marita Scimeca has recently begun practice in Kiowa, Kansas. Since her graduation in 1951, Doctor Scimeca has been practicing with her father in his office in Iola.

Some 350 persons joined a community party honoring Dr. C. E. Burtch of Portis, Kansas, as he completed his fiftieth year of practice in the medical profession.

Doctors V. E. Chesky, William C. Dreese, and C. A. Hellwig of Halstead presented a scientific exhibit on "Colloidophagy in the Thyroid Gland" during the June meeting of the American Medical Association in San Francisco.

Dr. C. T. McCoy has recently opened an office in Hutchinson.

At the recent meeting of the American Medical Association in San Francisco, Dr. Charles Pokorny of Halstead read a paper on "Diffuse Interstitial Fibrosis of the Lungs."

Dr. Carter B. Sigel has recently reported for duty in the U. S. Navy at the Naval Hospital, Oakland, California.

A brief note from Dr. Mary Glassen of Phillipsburg arrived from Geneva, Switzerland, where she attended the International Congress of Gynecology and Obstetrics, July 26-31. She says, "It is a brand-new experience for me to use the earphones constantly for interpretation of the four languages officially used. Naturally, I'm unreasonably proud of the U. S. participants. . . ." She also attended a meeting of the World Health Organization and closes



How to control itching and scaling for **1** *to* **4** *weeks*

You can expect results like these with SELSUN: complete control in 81 to 87 per cent of all seborrheic dermatitis cases, and in 92 to 95 per cent of common dandruff cases. SELSUN keeps the scalp free of scales for *one to four weeks*—relieves itching and burning after only two or three applications.

Your patients just add SELSUN to their regular hair-washing routine. No messy ointments . . . no bedtime rituals . . . no disagreeable odors. SELSUN leaves the hair and scalp clean and easy to manage.

Available in 4-fluidounce bottles, SELSUN is ethically promoted and dispensed only on your prescription. Abbott

prescribe

SELSUN®

Sulfide Suspension

(*Selenium Sulfide, Abbott*)

ACCEPTED COUNCIL ON PHARMACY AND CHEMISTRY AMERICAN MEDICAL ASSOCIATION

1-82-54

by saying that Europe has been wonderfully cool with Geneva almost too cool for comfort.

Dr. R. G. Rate, formerly of Veteran's Administration Hospital in Kansas City, has joined the staff at Hertzler Clinic in Halstead as head of the Department of Thoracic Surgery.

Dr. William J. Smith and family left for Warrensburg, Missouri, where his family will remain during Doctor Smith's tour of duty in the U. S. Navy.

Dr. Arthur L. Duell who was previously associated with the Gelvin-Haughey Clinic at Concordia, has recently opened an office in Clifton, Kansas.

Dr. Karl Menninger and five staff members of the Menninger Foundation have recently completed participation in a series of International Meetings on Mental Health in Toronto, Canada.

Dr. J. G. Claypool who recently left Howard, Kansas, to complete a residency in Internal Medicine, presented an exhibit at the June meeting of the American Medical Association in San Francisco.

Dr. Edward M. Burrell, began his medical practice in Turon, Kansas, on August 1, 1954.

Dr. Ernest P. Carreau has recently been released from active duty with the U. S. Navy and has recently made his residence at 461 South Glendale Street, Wichita, Kansas.

Dr. Porter E. Barbera has reported to the Brook Army Medical Center at Fort Sam Houston, Texas, to begin a two-year tour of duty in the U. S. Army.

Dr. Edward Maier has left Arkansas City for Rochester, Minnesota, where he has accepted a three-year fellowship with the Mayo Clinic.

ANNOUNCEMENTS

The Omaha Mid-West Clinical Society will hold its 22nd Annual Clinical Assembly, October 25-28, at the Hotel Paxton, Omaha, Nebraska.

The program includes guest and member speakers who will present addresses, clinics, panel discussions, question and answer periods, as well as other interesting features.

For further information, write the executive office of the Society, 1031 Medical Arts Building, Omaha, Nebraska.

Heart Meeting Canceled

Dr. P. W. Morgan, Chairman of the Committee on the Study of Heart Disease, announced that the 1954 annual postgraduate course in cardiology which was to be held at Emporia, Kansas, in October has been canceled. Plans for the 1955 meeting are now in progress.

The Kansas Medical Society Annual Meeting

The annual meeting of The Kansas Medical Society will be held at Hutchinson, Kansas, May 1-5, 1955.

Clinical Session of the A.M.A.

The mid-winter meeting of the American Medical Association will be held at Miami, Florida, November 29 through December 3, 1954.

Annual Meeting of the A.M.A.

The annual meeting of the American Medical Association will be held in Atlantic City, N. J., June 6-10, 1955.

40th Annual Clinical Congress of the American College of Surgeons

The 40th annual Clinical Congress of the American College of Surgeons will be held in Atlantic City, N. J., November 15-19, 1954.

Mind is the great lever of all things; human thought is the process by which human ends are ultimately answered.—*Daniel Webster*



Thank you doctor
for telling mother about...



The Best Tasting Aspirin you can prescribe

The Flavor Remains Stable down to the last tablet

Bottle of 24 tablets 15¢ (2½ grs. each)

We will be pleased to send samples on request

THE BAYER COMPANY DIVISION of Sterling Drug Inc., 1450 Broadway, New York 18, N. Y.

The Kansas State Board of Medical Registration and Examination at its regular session passed a resolution on June 9, 1954 stating that National Board Certificates issued on the basis of examinations, as presently conducted, would no longer be recognized as endorsement for licensure in this state.

COUNTY SOCIETIES

The Sedgwick County Medical Society has completed the construction of an executive office building and is now housed in new quarters at 1102 South Hillside, Wichita, Kansas. The telephone number also has changed to 63-7557.

This beautiful structure not only houses the executive office in air conditioned comfort, but contains their medical library and an attractive auditorium which is to be used for society and for public meetings of interest to medicine. Congratulations to the progressive society for another project successfully concluded.

BLUE SHIELD

The Doctor's Voice in Blue Shield

Comments received from physicians during the recent charge survey prompt us to again point out the doctor's voice in his "Health Plan," Blue Shield.

The Blue Shield Plan which Kansas doctors are concerned with, does and must have the approval of the Kansas Medical Society. The president and president-elect of the Society, along with a member from each Medical Councilor District, are on the Blue Shield Board. With the exception of three lay persons, the Board is composed entirely of Doctors of Medicine.

The Blue Shield Board of Trustees has supervision and control of the business, property, affairs and management of the corporation.

In addition to having control of the Board, the Kansas Medical Society appoints and controls two committees whose functions are of prime importance to Blue Shield operations. They are the Blue Shield Fee Committee and the Blue Shield Relations Committee.

The Blue Shield Fee Committee is composed of a representative from each of the medical specialities. It is the duty of this committee to accumulate and evaluate all suggestions and complaints from Participating Physicians and to then make recommendations to the Board for changes or additions to the Blue Shield Schedule of Allowances.

Any time a doctor participating in Blue Shield is dissatisfied with the Schedule of Allowances, he should make his feelings known to his representative on the Fee Committee.

Each of the Medical Councilor Districts provides a representative to make up the Blue Shield Relations Committee. These Councilor District representatives become chairmen of the District Relations Committees and each appoints a member from his County Society to round out his committee.

A staff member from Blue Shield meets with these District Relations Committees once or twice each year to discuss matters of policy, financial condition or any anticipated changes in the program. Through this means, each County Society's views are made known.

At this point, there should be little doubt as to the doctor's voice and the channels through which it can be expressed. If the individual doctor feels he has been by-passed in the process of policy making, perhaps it is because he hasn't made his wants known through the proper committee, or has assumed the attitude that his views are unimportant.

With the interested participation of all Society members, Blue Shield will prosper and remain truly the "Doctor's Health Plan."

Annual Fall Conference
Kansas City Southwest Clinical Society

The 32nd Annual Fall Clinical Conference of the Kansas City Southwest Clinical Society will be held in the Municipal Auditorium, Kansas City, Missouri, October 4, 5, 6, 7, 1954.

In addition to scientific lectures by the sixteen distinguished guest speakers, they will take part in seminars, conferences, and panel discussions, as well as the daily round table luncheon question periods.

The colored television program will be presented on the mornings of October 5, 6, and 7; many with guest speaker participants. This feature of the conference will include Operative Clinics, Clinical Demonstrations, and How You Do Its.

Your September-October copy of the *Kansas City Medical Journal* will reach your desk about the middle of September. Refer to it for the complete program of the Conference and plan to attend.

NOTICE

Members having material of historical interest relative to medicine or physicians of Kansas, please send it to the:

Committee on History
The Kansas Medical Society
315 West Fourth Street
Topeka, Kansas

record time

ROMYCIN*

Hydrochloride
Tetracycline HCl Lederle

ACHROMYCIN, new broad-spectrum antibiotic, has set an unusual record for rapid acceptance by physicians throughout the country. Within a few months of its introduction, ACHROMYCIN is being widely used in private practice, hospitals and clinics. A number of successful clinical tests have now been completed and are being reported.

ACHROMYCIN has true broad-spectrum activity, effective against Gram-positive and Gram-negative organisms, as well as virus-like and mixed infections.

ACHROMYCIN has notable stability, provides prompt diffusion in body tissues and fluids.

ACHROMYCIN has the advantage of minimal side reactions.

LEDERLE LABORATORIES DIVISION *AMERICAN Cyanamid COMPANY* Pearl River, New York

Lederle

*REG. U.S. PAT. OFF.

Adverse Effects of Penicillin Therapy

W. D. Bradshaw, M.D.*

Penicillin entered the armamentarium of the medical profession with dramatic and startling effectiveness in the early 1940's marking the beginning of what has been termed the "Era of Antibiotics." Many other antibiotics have been developed and are in wide use today; however, penicillin continues to be the most widely used chemotherapeutic agent. Why has penicillin gained such widespread usage? Perhaps the answer to this question is to be found in the following thoughts: (1) penicillin is truly a wonder drug having real chemotherapeutic value; (2) the enthusiasm and insistence of the general public for penicillin therapy; and (3) the "memory" of the medical profession of the pre-antibiotic era when infections which seem insignificant now were often overwhelming and fatal.

One of the first reports of the clinical use of penicillin, published in March of 1943 by the Floreys,[1] made no mention of undesirable effects. In August of 1943[2] reports of untoward reactions began to appear in medical literature. During the past eleven years a voluminous amount of material has been published concerning adverse effects of penicillin therapy.

It is of importance to attain a workable knowledge of these adverse effects in order to realize the fullest therapeutic value of this agent. A clinical evaluation of penicillin is still far from complete, but some perspective concerning its present status is possible and in order at this time. The adverse effects produced by penicillin may be grouped and will be discussed as (1) hypersensitivity reactions, (2) effects related to bacterial alterations, (3) direct toxic reactions, and (4) effects on patient care.

EFFECTS RELATED TO HYPERSENSITIVITY TO PENICILLIN

Hypersensitivity or allergy to penicillin has received wide publicity in the past decade. Despite the fact that many competent people have and are investigating this problem, there remains much to be desired for adequate understanding. It must be remembered that our commercial penicillins are derived from a common mold which is known to be antigenic and which is widely distributed in nature.

Incidence: The incidence of penicillin allergy in the general population is extremely difficult to evaluate. Rhodes[3] reports an incidence of three to four per cent; Long,[4] an incidence of five per cent. It will be noted that the incidence of some small group or particular type of reaction reported in this paper will exceed the incidence which Long reports; a fact which illustrates the varying opinions expressed in the current literature as to incidence of these manifestations. It is interesting to note that most of the allergic manifestations reported in the literature have been in the adult population. Apparently these reactions are not common in pediatrics although such occur in children.

Immunology: Little is known concerning the basic immunology of the reactions. Most of the ordinary rules that one might expect to apply seem to be remarkably unreliable in relation to penicillin reactions. Penicillin combines with human serum albumin as shown by dialysis experiments; it is believed to possess antibiotic activity when in such combinations.[5] This fact might tend to make one think of penicillin as a haptene; however, such has not been proven.

Route of administration: The route of administration of penicillin is of consequence in the consideration of various hypersensitivity phenomena.

Topical application is commonly recognized today as a cause of contact dermatitis.[6] Sensitization is usually developed in the course of one to two weeks after the first contact with penicillin ointment. Allergic reactions have been reported from medical personnel handling penicillin.[7, 8, 9] Templeton, et al., in 1947,[10] reviewed the toxic reactions occurring after the topical application of penicillin solutions and ointments. They recommend that topical use be avoided; this recommendation has apparently been well accepted by the medical profession.

Local applications of penicillin in the form of lozenges and drops may cause local reactions such as apthous stomatitis, rhinitis, glossitis, and melanoglossia.[11, 12] The reaction may not necessarily be local, but may, in a highly sensitive individual, produce a generalized sensitivity reaction, even when penicillin is used in oral lozenge form.[13] Mayer, et al.[14] states that the incorporation of penicillin in gums, gargles, and mouth and throat lozenges should be condemned. Because of the increased potential of sensitization this mode of administration should be avoided and systemic medication substituted for greater efficacy.

Intrathecal administration of penicillin is not dangerous because of the sensitivity factors but rather as a direct toxin to the central nervous system. Attention

---

* This is one of the theses written by fourth year students in the University of Kansas School of Medicine selected for publication by the Editorial Board from a group judged by the faculty at the school to be the best.

Thesis written while the author was a fourth year student. Dr. Bradshaw is now interning at the University of Kansas Medical Center, Kansas City, Kansas.

VERILOID®

A POTENT, NOTABLY SAFE HYPOTENSIVE

Veriloid, the alkavervir extract of the hypotensive principles fractionated from Veratrum viride, presents these desirable properties in the management of hypertension.

- Uniform potency and constant pharmacologic action assured by biologic assay . .
- Blood pressure lowered by centrally mediated action; no ganglionic or adrenergic blocking, therefore virtually no risk of postural hypotension . .
- Cardiac output not reduced; no tachycardia . .
- Cerebral blood flow not decreased . .
- Renal function unaffected . .
- Tolerance or idiosyncrasy rarely develops; hence Veriloid is well suited to long-term use in severe hypertension . .
- Notably safe . . . no dangerous toxic effects . . . no deaths attributed to Veriloid have been reported in over five years of broad use in literally hundreds of thousands of patients . .
- Side actions of sialorrhea, substernal burning, nausea and vomiting (due to overdosage) are readily overcome and avoided by dosage adjustment.



TABLETS VERILOID

Supplied in 2 mg. and 3 mg. slow-dissolving scored tablets, in bottles of 100. Initial daily dosage, 8 or 9 mg., given in divided doses, not less than 4 hours apart, preferably after meals.



SOLUTION INTRAVENOUS

For prompt reduction of critically elevated blood pressure in hypertensive emergencies. Extent of reduction is directly within the physician's control. In boxes of six 5 cc. ampuls with complete instructions.



SOLUTION INTRAMUSCULAR

For maintenance of reduced blood pressure in critical instances, and for primary use in less urgent situations. Single dose reaches maximum hypotensive effect in 60 to 90 minutes, lasts 3 to 6 hours. Boxes of six 2 cc. ampuls with complete instructions.

Riker LABORATORIES, INC. Los Angeles 48, California

will be given to this route of administration of penicillin as a direct toxic agent.

Keefer[15] states that the greatest incidence of sensitization arises from topical administration and the least from the oral route. Finland[16] states that although oral penicillin is only about 10 to 30 per cent as effective as intramuscular administration it has a relatively low sensitizing effect and can be used in sensitive individuals. He also states that the soluble forms have much less sensitizing potential than do the repository forms.

Types of penicillin: The large scale production of hypoallergic penicillins bears witness to the fact that penicillin reactions are attaining an incidence higher than the public or the medical profession will tolerate without comment. To understand the various types of penicillin, a brief statement in regard to the preparation of these compounds seems pertinent.

Penicillin is a generic term and designates a group of compounds that contain an alanine dimethyl cysteine nucleus but which differ in the side chain composition. A variety of "substituted penicillins" or "biosynthetic penicillins" can be prepared by incorporating the appropriate precursors in the media in which Penicillium Notatum is grown.[17]

Penicillin "G," characterized by a benzyl group in the side chain, is the most widely used commercial penicillin. Penicillin "O" or "AT" has a straight chain replacing the benzyl ring of penicillin "G"; its chemical term is allylmercaptomethyl penicillin. Penicillin "BT" also has a straight side chain; chemically termed N-butylthionethyl penicillin. "Compenamine" is 1-ephenamine penicillin "G." The claim of lesser potential of sensitization has been made for this preparation as has been made for penicillin "O" and "BT."

It has been demonstrated that there is considerable cross-sensitization between penicillin "G," "O," and "BT."[17] However, not all individuals show sensitivity to all such perparations; therefore, the usefulness of the different forms of penicillin should not be overlooked, since it might be possible to substitute the various forms if so required. Compenamine deserves further investigation before a true evaluation may be discussed.

Although chemical precursors may be fed to microorganisms producing penicillin in an attempt to produce a drug that is not normally produced or to overcome the sensitization problem, nature cannot be made to forget completely her natural manufacturing methods. Hence, according to the current information, penicillins BT and O probably contain a small amount of penicillin G as a contaminant. In general, the hypoallergic penicillins have not proven to be as useful as was anticipated.

Penicillin preparations combined with antihistamics have been used; this attempt to reduce hypersensitivity phenomena appears to have some merit. More study is needed for complete evaluation of this method of therapy.

A constant effort has been made since penicillin first became available to alter the molecule in such a manner as to enhance certain valuable properties. What effect do these additions to the penicillin molecule have on hypersensitivity phenomena? Penicillin in beeswax and oil produced an unusual incidence of the urticarial type of reaction especially at the site of injection. The oil and wax probably act as adjuvants increasing the tendency to sensitization in addition to delaying the absorption of antigens, thus producing a more severe local reaction.[19] There has been no conclusive proof that procaine has occasioned any additional sensitization, although it should be noted that sensitivity to procaine itself is a real possibility. Penethamate ("Neo-penil") has been shown to diffuse more readily into lymph glands, the respiratory tract, and central nervous system than penicillin G. However, serious side effects have been reported recently, rendering this product of questionable usefulness. Benzethasil ("Bicillin" or "Permapen") represents a modification of penicillin designed to produce prolonged plasma concentrations. There is no evidence incriminating this preparation as being more sensitizing than any other preparation. All of the preparations of penicillin representing chemical unions of two or more drugs must be evaluated carefully because the potentials of sensitization may be increased over the potential of penicillin alone.[20]

Biologic tests for hypersensitivity: Attempts to determine hypersensitive individuals by various biological tests are of interest. Procedures which are most frequently used in clinical medicine include the scratch, intracutaneous, patch, and inhalation tests.

The skin tests are conducted in the manner of the usual routine testing for hypersensitivities. The clinical significance is, as yet, open to some question; however, the tests do serve to call attention to possible disastrous effects that may follow penicillin administration. The following facts appear to be apropos:

1. Negative skin test does not rule out the possibility of a serious reaction.

2. A positive test in an individual with a history of previous penicillin reactions or a history of having had one or more courses of penicillin is certainly indicative of clinical sensitivity and the likelihood of a serious reaction should be considered.

3. Patients showing a delayed positive type of reaction to skin tests are probably not as likely to have immediate reactions as the more common urticarial or "serum-sickness" type of reaction.

4. A strong, immediate urticarial reaction to skin tests indicates a very unusual degree of sensitivity

KARO SYRUP

BELONGS IN THIS PICTURE!

...a carbohydrate of choice
in milk modification for 3 generations

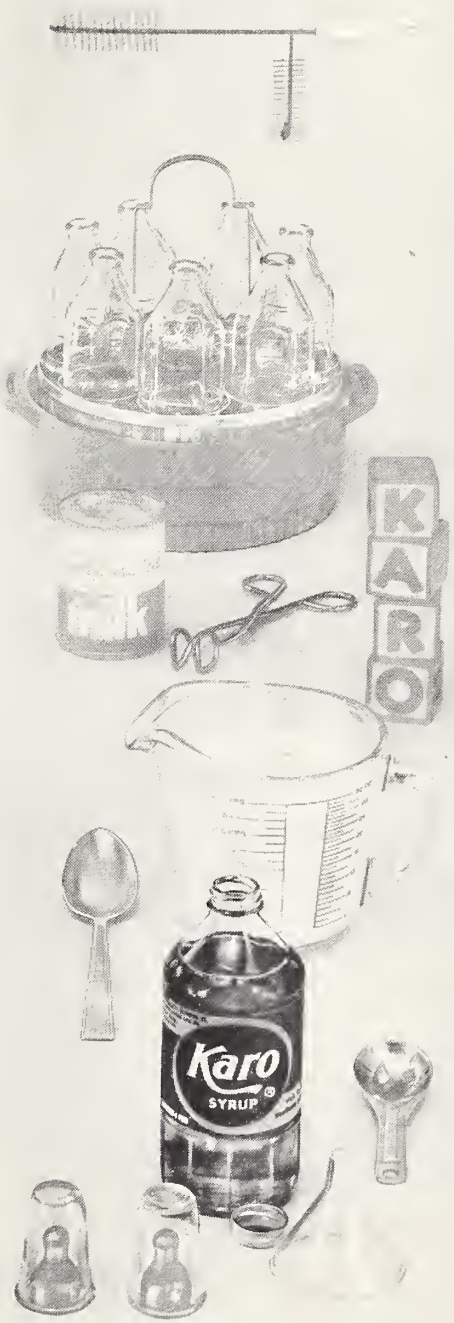
OPTIMUM caloric balance—60% of caloric intake, gradually achieved in easily assimilable carbohydrates—is assured with Karo. Milk alone provides 28%, or less than half the required carbohydrate intake.

A MISCIBLE liquid, Karo is quickly dissolved, easy to use, readily available and inexpensive.

A BALANCED mixture of dextrins, maltose and dextrose, Karo is well tolerated, easily digested, gradually absorbed at spaced intervals and completely utilized.

PRECLUDES fermentation and irritation. Produces no reactions, hypoallergenic. Bacteria-free Karo is safe for feeding prematures, newborns, and infants—well and sick

LIGHT and dark Karo are interchangeable in formulas; both yield 60 calories per tablespoon.



CORN PRODUCTS REFINING COMPANY
17 Battery Place, New York 4, N. Y.



and the greatest possible danger if ordinary doses of penicillin are given.

5. The relative merits of the patch, intracutaneous, and scratch tests is much debated. It appears that the patch test is of questionable value.[21] Severe hypersensitive generalized reactions have occurred following intracutaneous tests.[22] Patients suspected of having a high degree of sensitivity should be tested first by the scratch method and then, if negative, by the intracutaneous method.

Inhalation tests using penicillin as an areosol have been used to determine the reactivity of the bronchial tree or lung. Measurements of vital capacity are recorded as the allergen is inhaled.[23] This test has not been widely used and deserves more study for evaluation.

Several other biological tests are available but have not received as much clinical use as the skin tests. The methods that have been used are passive transfer or passive sensitization of human skin, the Dale reaction in guinea pigs, and precipitin tests for antibody production. These largely represent experimental studies designed to gain an insight into the basic immunology of these penicillin reactions.

Desensitization: Desensitization of individuals to penicillin has been attempted. Peck and Seigal[24] report a case of successful desensitization. However, such procedures have not been uniformly successful and deserve more study and evaluation.

Relation of penicillin to collagen diseases: Is there a correlation between penicillin hypersensitivity and the "collagen diseases"? Rich[25] showed a relationship of the hypersensitivity state to periarteritis nodosa, lupus erythematosis, serum-sickness, and rheumatic fever. A report by Walsh and Zimmerman[26] of six patients with penicillin reactions provides interesting speculation as to a relationship between lupus erythematosis and hypersensitivity. Three of their patients having severe penicillin reactions showed the "L.E." phenomenon and in the three with less severe reaction, the cells were not found. The authors draw no conclusions as to the role of penicillin in these cases. The question of a correlation between penicillin and the collagen diseases is indeed an interesting problem.

The commonly observed types of hypersensitivities to penicillin will be considered under the major groupings of dermatologic effects, serum-sickness-like reactions, immediate constitutional reactions, and other less common manifestations.

DERMATOLOGIC EFFECTS

The early reports of dermatological reactions were concerned only with urticaria, the incidence of which ranged between two to three per cent.[27] During the years of increasing penicillin usage, dermatological reactions have increased in variety and in frequency. Current literature includes epidermophytid-like lesions, dermatitis medicamentosa, contact dermatitis, the various urticarial groups, and many other dermatological descriptive lesions. In 1950, Long[4] estimated the incidence of dermatological reactions to be from 10 to 15 per cent, illustrating the greater frequency as more people are being exposed to penicillin. However, it must be realized that penicillin reactions are being recognized with greater frequency since more knowledge has accumulated concerning them.

A workable understanding of these reactions is probably best attained by a consideration of their pathogenesis and physical appearance. Reyer[29] organizes these reactions from the standpoint of their pathogenesis as follows:

Group A: Epidermal contact reactions with localized dermatitis and sensitization restricted to areas of application.

Group B: Dermal reactions regardless of the route of administration, whether by local application, by ingestion, by inhalation, or by injection.

Type I: Specific penicillin reactions.

Type II: Reactions to the vehicle, the base, or the culture medium upon which the penicillin is grown.

Type III: Nonspecific reactions to the common mold antigen.

Type IV: Jarish-Herxheimer reactions of the bacterial type from liberation of toxins from organisms killed by penicillin.

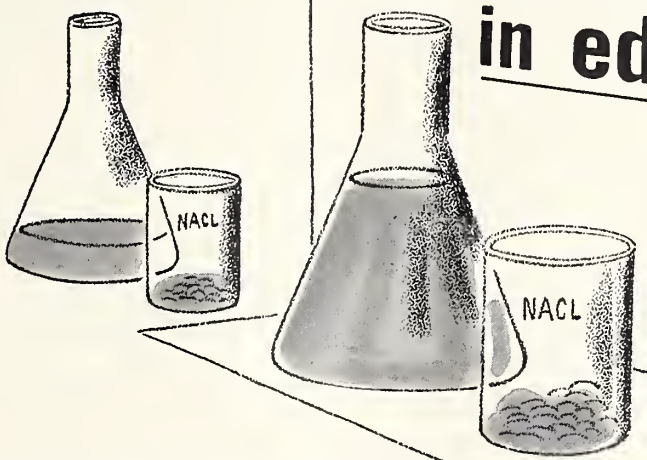
Type V: Reactions from a disturbance of the "symbiotic balance" of organisms growing on the skin surface.

A more complete view of the problem is gained by considering each of these mechanisms with the physical appearance and characteristics of the reactions.

Epidermal contact reactions: The epidermal contact reaction is a typical vesicular contact dermatitis. Sensitization usually develops in the course of one to two weeks after first contact with penicillin ointments. External contact may well cause a generalized dermal sensitization.[10] Because of the high index of sensitization as well as the possibility of inducing a generalized dermal sensitization, local use of this antibiotic has been condemned.

Dermal reactions specific to penicillin: Dermal reactions specific to penicillin consist primarily of the urticarial reactions. This reaction is a delayed type, coming on seven to twenty one days after the first administration of the drug and apparently is induced by treatment. This delayed incubation period may be shortened as an accelerated reaction seen in cases receiving a second or third exposure to penicillin. It appears to be a common belief that these urticarial reactions are easily controlled and may be of

increasing the excretion of water and sodium in edema



The aim of edema therapy is twofold: to increase the volume of fluid excreted from the body and, of equal importance, to effect a removal of water-binding sodium ions.

Salyrgan-Theophylline, established through the years as a dependable mercurial diuretic, performs both of these functions.

SUPPLIED:

Ampuls of 1 cc. and 2 cc. — boxes of 10, 25 and 100.

Tablets — bottles of 100, 500 and 1000.

SALYRGAN®-THEOPHYLLINE

Clinical response to Salyrgan-Theophylline is usually rapid. Within the first day after administration much of the excess tissue fluid is mobilized and eliminated. Up to 10 liters may be excreted in a twenty-four hour period. Similarly, excretion of 20 Gm. or more of sodium chloride within twenty-four hours after Salyrgan-Theophylline has been observed.[1,2]

For removal of edema and ascites in cardiac and cardiorenal diseases; nephrosis, and cirrhosis of the liver.

Salyrgan, brand of mersalyl, trademark reg. U. S. Pat. Off.

WINTHROP

Winthrop-Stearns INC.
NEW YORK 18, N. Y. WINDSOR, ONT.

1. Nielsen, A. L., Bechgaard, P., and Bong, H. O.: Low-Solt Diet in Treatment of Congestive Heart Failure. *Brit. Med. Jour.*, 1:1349, June 16, 1951.

2. Brown, W. E., and Sutherland, C. G.: Control of Edema in Pregnancy. *GP*, 8:65, Nov., 1953.

little consequence; however, Epstein[22] reports six patients in whom the urticaria persisted for from three to fifteen months. Each of the six patients remarked independently that he would rather have the infection for which the penicillin was given.

The urticarial type reaction of severe nature and associated with serum-sickness-like signs and symptoms will be discussed later.

Reactions to the vehicle: Many patients' case histories appearing in the literature do not mention testing for hypersensitivity to the vehicle, the base, the culture medium used to grow the penicillin, or to other brands of penicillin. It is, therefore, difficult to separate those reactions due to the penicillin itself from those due to the other agents. No characteristic physical appearance can be set forth for this group as the manifestations may vary with the responsible agent.

Non-specific reactions to the common mold antigen: This type of mechanism is probably responsible for most of the immediate eczematous, bullous, or vesicular eruptions often described. This type of reaction is frequently referred to as the "id" type of reaction because, at least superficially, it often resembles an attack of epidermophytosis involving particularly the interdigital spaces, the groin, and flexion creases. The characteristic time of occurrence is early with an incubation period of a few hours to not more than one day. Lesions are usually observed to be red, oozing, and itching. These reactions may also be of a severe nature; Huber[22] reported an exfoliative dermatitis following penicillin in a patient with a chronic epidermophyte infection about the feet of several years' duration. Twelve hours after injection of penicillin a generalized papulopustular eruption appeared, and on the sixth day the skin over the entire body exfoliated.

The Jarish-Herxheimer reactions: These dermal reactions are not due to penicillin sensitivity but are rather due to a sensitivity to the products of decomposition of bacteria or fungi. The bacteria or fungi that liberate the products must be nonresistant to penicillin, thus flooding the system with the toxins of the disintegrating organisms. This type of reaction is commonly observed to be of the eczematous type. Systemic reactions are of frequent occurrence in addition to the skin manifestations.

Symbiotic imbalance: The effect of penicillin in altering normal flora of the host is certainly a complex problem. Skin organisms of course share in this disturbance as do the micro-organisms in other parts of the body. Many cases listed as allergic eczematous eruptions, id eruptions, or flareups of fungal infections may ultimately be traced to such imbalances of skin organisms brought about by the sudden presence of penicillin. This interesting concept will be discussed further as "superinfections."

DELAYED "SERUM-SICKNESS-LIKE" TYPE OF REACTION

The term "serum-sickness" type of reaction appears to be a very applicable term because this type of reaction can imitate practically all of the usual features of a reaction to heterologous serum. This is probably the commonest type of hypersensitivity to penicillin. The incidence of these reactions as reported in the literature varies from one per cent[20] to six per cent[14] of all patients receiving penicillin.

Cecil[18] defines serum-sickness as "an allergic reaction resulting from the parenteral administration of a foreign serum and characterized by an incubation period, skin eruption, enlargement of lymph nodes, fever, edema, and polyarthritis."

Like serum-sickness this type of reaction has an incubation period of about five to fourteen days. This delayed reaction time and incubation period probably indicates that the patient is developing sensitivity as a result of administration of the drug.

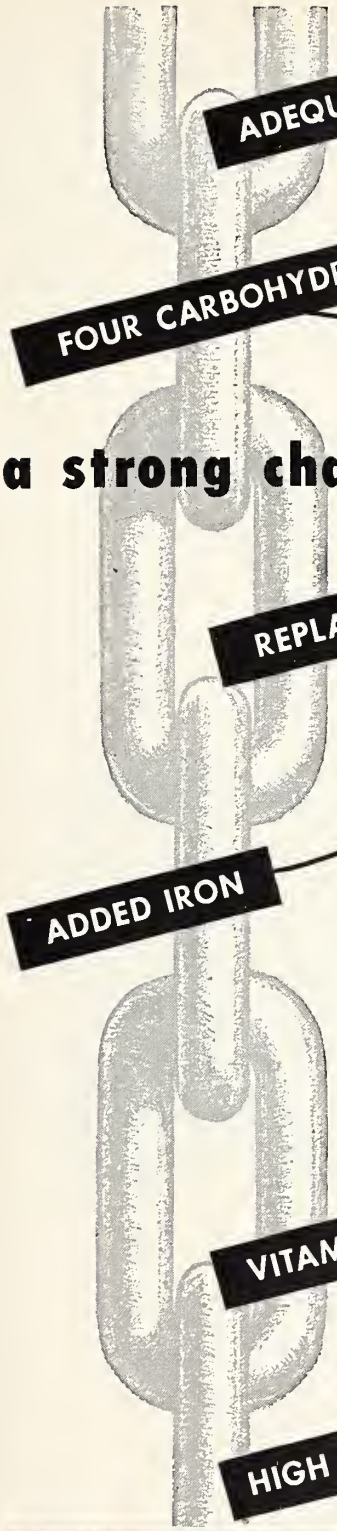
Intramuscular injections of the drug have been reported as the most frequent mode of administration although these reactions occasionally follow oral therapy. In many instances previous local applications have been the most likely cause of the hypersensitivity. Riley[28] reporting twenty-two cases of serum-sickness from penicillin states that eighteen of the twenty-two previously used penicillin locally.

The predominant feature is urticaria and angioneurotic edema. The palms of the hands and soles of the feet are characteristically involved with the urticaria. Severity of this reaction varies, as it does with serum-sickness, from a relatively mild and brief urticaria to an extremely severe illness which may last for several weeks. There is often an arthalgia and swelling of the joints, particularly the small joints of the hands and feet. Fever is a variable finding. It may be absent or negligible in mild cases or it may be as high as 105° in more severe cases. Patients presenting themselves complain bitterly of severe pruritis which usually begins just prior to the urticarial formation and may continue in the face of therapy.

Gastro-intestinal symptoms may or may not be associated with this reaction. The symptoms most often described are nausea and vomiting with accompanying anorexia. Diarrhea is infrequently a complaint.

Psychogenic symptoms are often marked. Apprehension of striking magnitude is frequently observed. Toxic psychosis appearing in a patient reported by Kline and Highsmith[30] was thought to be due to edematous lesions in the brain resulting from a localized penicillin hypersensitivity. However, the usual symptoms seen are those of severe apprehension often accompanied by moderate to severe malaise.

The duration of this reaction is extremely variable, ranging from four or five days to eight weeks or



ADEQUATE PROTEIN

When fed as suggested, Baker's Modified Milk supplies 3.7 grams of protein per kilogram of body weight per day.

FOUR CARBOHYDRATES

In normal dilution, Baker's Modified Milk contains 7% carbohydrate in the form of lactose, dextrins, maltose and dextrose.

a strong chain is made from strong links

REPLACED FAT

The butterfat is replaced by a select combination of vegetable and animal fats to provide 85% of the fat composition in the more readily digestible range.

ADDED IRON

Iron is added to provide 7.5 mg. per quart.

Baker's Modified Milk

FOR BOTTLE-FED INFANTS

VITAMIN FORTIFIED

Each quart of Baker's contains 2500 U.S.P. units Vitamin A; 800 U.S.P. units Vitamin D; 50 mgms Ascorbic Acid (C); 0.6 mgm Thiamine; 5 mgms Niacin; 1 mgm Riboflavin; 0.16 mgm Vitamin B_6.

HIGH QUALITY MILK

Made from Grade A Milk (U.S. Public Health Service Milk code), modified as described above.

BAKER'S MODIFIED MILK

THE BAKER LABORATORIES INC.

Milk Products Exclusively for the Medical Profession

Main Office: Cleveland 3, Ohio
Plant: East Troy, Wisconsin

Division Offices: Atlanta, Dallas, Denver, Greensboro, N. C., Las Angeles, San Francisco, Seattle

ACCEPTED COUNCIL ON FOODS AND NUTRITION AMERICAN MEDICAL ASSOCIATION

more. It is important that these patients be informed that the antigenic substance will, in time, be excreted or changed in such a manner that the reaction will subside.

SEVERE IMMEDIATE CONSTITUTIONAL REACTIONS

Although unfavorable reactions to penicillin therapy are well known, the occurrence and frequency of serious, life-threatening, immediate constitutional reactions have only recently been generally recognized. The incidence of these severe reactions is at best an approximation and probably is impossible to calculate because only a fraction of such reactions are publicized.[31] It seems that this problem is becoming a more frequent one. During the 15 month period preceding February, 1953, there were 15 cases of penicillin anaphylaxis published and, of these, six were fatal. By contrast from 1946 to 1951, there were only six cases reported with but one fatality.[20] It is almost universally accepted that the number of penicillin sensitive persons is increasing with widening use of this powerful drug. This statement or similar ones are repeatedly found throughout the current literature.

The immediate constitutional reactions to penicillin present a fairly characteristic picture. The onset is rapid, ranging from a few seconds to several minutes after administration of the drug. Complaints frequently offered by patients include: strange taste sensations, itching, a feeling of compression of the chest, feelings of impending death, difficult breathing, weakness and extreme nervousness. The most important objective signs are those of shock, with a profound fall in blood pressure, labored slow breathing, and cyanosis. Death occurs within the space of a few minutes to two or more hours and appears to result from shock or asphyxia.

Starling[32] sets forth five cardinal symptoms which serve as a forewarning of this type of reaction. These symptoms are: (1) syncope, mild to severe, at the time of injection, or a feeling of faintness recurring periodically for the following three to four hours; (2) sensation of burning or heat throughout the body, especially in the throat, palms of hands, or soles of the feet (similar to reaction following intravenous calcium); (3) local or generalized pruritis, never before present, may occur in various parts of the body, beginning within a few hours after injection; (4) choking sensation in the throat, with increasing cough, dyspnea, and wheezing; (5) severe paroxysmal pain varying in location, such as precordial, epigastric, or diaphragmatic.

There is no predictable pattern in penicillin hypersensitivity reactions; several courses of penicillin may not give rise to symptoms before and even after severe allergic reactions.[33]

The route of administration which has most commonly been reported in association with these severe reactions is intramuscular. It should be realized, however, that accidental intravenous injection may have been responsible for many such reported reactions. These hypersensitivity phenomena are not restricted to the intramuscular route; severe reactions have been described from intranasal instillation,[34] aerosol,[35] oral administration,[36] and intracutaneous skin tests.[37] Information at the present time does not incriminate any one single preparation of penicillin. It is possible that some of these reactions may be due to a hypersensitivity to procaine; however, one published fatality[38] can be blamed on the fact that the author reporting it was led to believe that a previous anaphylactic reaction in that patient was due to the procaine rather than the penicillin.

Pathological studies indicate that changes likely to be found are emphysema, edema in the lungs and bronchi, congestion of the lungs with small hemorrhages, and congestion of the spleen and kidneys. In some instances there may be very little of significance

TABLE 1: REACTIONS FOLLOWING PENICILLIN THERAPY

Reaction	*Bibliography*
Peripheral neuritis	JAMA 132:323 (Oct. 12) 1946.
Dermographia	Arch. Derm. & Syph. 54:66 (July) 1946.
Delirium	Brit. M.J. 1:240 (Feb. 5) 1949.
Mycocarditis, arteritis, and focal hepatic, splenic, and renal granulomata	Am. J. Path. 28:437 (May-June) 1952.
Purpura	Ann. Int. Med. 34:1219 (May) 1951.
Swollen tongue	Cincinnati J. Med. 28:321 (May) 1947.
Myocarditis	JAMA 143:361 (May 27) 1950.
Arthritis	Ibid.
Hepatitis	Ibid.
Loeffler's syndrome	Ann. Int. Med. 38:113 (Jan.) 1953.
Bronchial spasm	J. Mt. Sinai Hosp. 14:460 (Sept.-Oct.) 1947.
Ocular dermatitis	Arch. Ophth. 35:145 (Feb.) 1946.
Oral hemorrhage	Brit. M.J. 2:760 (Nov. 16) 1946.
Agranulocytosis	Ann. Int. Med. 25:732 (Oct.) 1946.
Asthma	JAMA 128:183 (May 19) 1945.
Eosinophilia-asymptomatic	Ann. Allergy 6:675 (Nov.-Dec.) 1948.
Hypoprothrombinemia	JAMA 140:1127 (July 30) 1949.
Cheilosis	Brit. M.J. 2:638 (Oct. 26) 1946.
Local hemorrhagic-necrotic skin reaction	J. Pediat. 38:630 (May) 1951.
Intestinal ulceration and hemorrhage with associated dermatologic reactions and vascular necrosis	New Eng. J. Med. 242:814 (May 25) 1950.
Anaphylactic purpura	M. J. Australia 34:305 (March 8) 1947.
Sterile abscess formation	JAMA 134:1475 (Aug. 23) 1947.

for greater safety in streptomycin therapy...

DISTRYCIN

Squibb Streptoduocin
Streptomycin and dihydrostreptomycin in equal parts

Distrycin has an important advantage over streptomycin. It has the same therapeutic effect but ototoxicity is greatly delayed. Since the patient is given only half as much of each form of streptomycin as he would have on a comparable regimen of either one prescribed separately, the danger of vestibular damage (from streptomycin) or cochlear damage (from dihydrostreptomycin) is significantly lessened.

Signs of vestibular damage appear in cats treated with Distrycin as much as 100 per cent later than in animals given the same amount of streptomycin.

Cat treated with streptomycin shows no nystagmus after whirling.



Cat given the same amount of Distrycin has normal reflex.



On dosage of 1 Gm. per day for 120 days, ototoxicity was as follows*:

	Vestibular damage % of patients		
	Mild	*Moderate*	*Total*
Streptomycin	12	6	18
Dihydrostreptomycin	6	0	6
Distrycin	0	0	0

	Cochlear damage % of patients		
	Mild	*Moderate*	*Total*
Streptomycin	0	0	0
Dihydrostreptomycin	12	3	15
Distrycin	0	0	0

*Heck, W.E.; Lynch, W.J., and Graves, H.L.: Acta oto-laryng. 43:416, 1953.

Distrycin dosage is the same as for streptomycin. In tuberculosis the routine dose is 1 Gm. twice weekly, in conjunction with daily para-aminosalicylic acid or Nydrazid (isoniazid). In the more serious forms of tuberculosis, Distrycin may be given daily, at least until the infection has been brought under control.

SQUIBB

a leader in streptomycin research and manufacture

'Distrycin'® and 'Nydrazid'® are Squibb trademarks

Distrycin is supplied in 1 and 5 Gm. vials, expressed as base

found at autopsy. Several observers have stated that post-mortem findings are often the "guinea pig anaphylactic lungs."

LESS COMMON REACTIONS

Many case reports of hypersensitivity to penicillin appear in the current literature which do not correspond to the previously considered common types. Although reactions in large variety have followed penicillin administration, some may, of course, not be due to penicillin.

Table I illustrates many of the unusual reactions following penicillin reactions. There is no intention to draw conclusions or discuss the findings but merely to point out the extreme variability of "penicillin reactions."

Undoubtedly many more unusual reactions following penicillin therapy have and will occur. Many such manifestations are probably not caused by penicillin, although some of the listed reactions appear to be well proven cases of penicillin hypersensitivities.

Prophylaxis of hypersensitivity reactions: Seigal[39] is of the opinion that with repeated exposure to penicillin through the years, largely by injection, increasing numbers of persons are becoming sensitized to the anaphylactic level.

"The era of mild acceptable nondisabling post-penicillin reactions is changing to include graver complications; guilt is in usage; penicillin is not a cure-all."[40]

Certain precautions are worthy of serious considerations before instituting treatment with penicillin:

1. Avoid the use of penicillin for trivial or doubtful indications.

2. Secure a careful history of previous penicillin treatment and all adverse effects. Previous reactions of any type are significant. Accelerated and especially immediate reactions are of extreme significance. If previous reactions are recognized, the best therapy is to use a different antibiotic having the same bacterial spectrum.

3. Oral penicillin is apparently less likely to cause the severe immediate constitutional reactions than is the parenteral route. Topical application is well known to be a frequent sensitizer and is best avoided.

4. The possibilities of activating an old fungus infection is real.

5. Patients having conditions which apparently will respond to penicillin only, and who are suspected of being hypersensitive to the drug, should have preliminary skin tests. An additional helpful precaution might well be the oral administration of a 50,000 unit penicillin tablet ten minutes before the intramuscular injection.

6. Waldbott[41] suggests that it may be wise to inject the first drop or two, then pause for forty-five seconds before completing the injection, as the really violent reactions may become evident within seconds.

7. An additional precaution is modification of the technique of injection; the initial or potentially shocking dose being given into the arm rather than into the buttock so that a tourniquet may be applied if necessary.

Of these precautions, the first is of prime importance. The use of "prophylactic penicillin" is of questionable usefulness except, perhaps, for those conditions for which it has been of well established value as in rheumatic fever. Penicillin is a specific drug, its use is to be sanctioned only when it is administered for specific chemotherapeusis.

Treatment of the hypersensitivity reactions: The treatment of these hypersensitivity reactions is symptomatic. Prompt institution of treatment as soon as a diagnosis is made will often result in remission. The antihistaminic drugs are of value in relieving the pruritis and severity of the urticarial lesions. Bed rest is valuable and sedation is often indicated in the apprehensive patient. The more severe forms of serum-sickness, severe dermatological reactions, and anaphylactoid reactions are often afforded relief by ACTH or cortisone. In the immediate anaphylactoid type of reaction, of course, the treatment is that of any anaphylactic reaction with epinephrine the main reliance.

Reports in the literature often point out that mild reactions may not interfere with the continuance of penicillin therapy. It seems to me that continuing penicillin therapy in the face of an allergic reaction, be it even the mildest reaction, is inviting trouble. With the development of newer antibiotics it appears reasonable to substitute another antibiotic for penicillin even though it may be somewhat less efficient rather than to take the chance of superimposing a disease more severe than that for which therapy was instituted.

EFFECTS RELATED TO BACTERIAL ALTERATION

It has been shown that adverse effects of penicillin therapy may be produced by changes in the host; adverse effects may also be produced by changes in the bacteria, altering the bacteria in such a manner that penicillin no longer can be used as a therapeutic tool.

Bacterial resistance: The mechanism of evolution in the biological kingdom is considered to be spontaneous mutation followed by natural selection or "survival of the fittest."

This problem of bacterial resistance to penicillin in clinical medicine has been largely concerning the staphylococci and the enterococcal streptococci. There has been no evidence of decreased activity *in vivo* of the more sensitive bacteria: gonococci, pneumococci, or beta hemolytic streptococci. Penicillin treatment of syphilis has not been affected by drug fastness.

As stated, staphylococci comprise the largest and

50 *and seven*

YEARS TREATING ALCOHOL AND DRUG ADDICTION

In 1897 Doctor B. B. Ralph developed methods of treating alcohol and narcotic addiction that, by the standards of the time, were conspicuous for success.

Twenty-five years ago experience had bettered the methods. Today with the advantages of collateral medicine, treatment is markedly further improved.

The Ralph Sanitarium provides personalized care in a quiet, homelike atmosphere. Dietetics, hydrotherapy and massage speed physical and emotional re-education. Cooperation with referring physicians. Write or phone.

The

RALPH

SANITARIUM

A Unit of the Benjamin Burroughs Ralph Foundation for Medical Research

Telephone VIctor 3624



529 HIGHLAND AVENUE • KANSAS CITY 6, MISSOURI

Ralph Emerson Duncan, M.D., Medical Director.

most important single group of resistant bacteria. It appears to be generally agreed that there are two kinds of resistance to penicillin in staphylococci: (1) One is that which can be induced artifically *in vitro* and was first described by Abraham et al.[42] in 1941. This type of resistance appears to be temporary in duration and exhibits morphological and Gram-staining changes and is accompanyed by a loss in virulence. (2) The other type of resistance is that which is known as "natural resistance," produced by virtue of the ability of the micro-organism to change its metabolism or to produce penicillinase and to retain full virulence for experimental animals.[43] The subject has been a widely controversial one. The production of penicillinase is one physiological mechanism of the bacteria to develope resistance, the one which has received the widest publicity. However, several other mechanisms may also be responsible for this resistance.[44]

Do staphylococci readily become resistant to penicillin *in vivo,* and if so, in what way does this resistance differ from the kind acquired *in vitro?* Barber's suggestion[45] is that what appears to be an increase in penicillin resistant staphylococci following or during therapy may really be the result of a selective process in which sensitive bacteria are destroyed, while resistant ones survive and proceed to multiply.

In a report of three cases of fatal staphylococcus enteritis occurring after prophylactic penicillin and dihydrostreptomycin, Fairlie and Kendall[46] point out that the concept that this complication is due to suppression of normal intestinal flora permitting pathogenic overgrowth of intestinal staphylococci appears to be oversimplified. A direct stimulation of the staphylococcus by antibiotics must be considered. This interesting idea needs more observation and study before it can be fully appreciated.

Attention to the prevalence of penicillin resistant staphylococci brings a recognition of a gradual elimination of sensitive strains. The increased resistance of these pathogens has been noted especially in hospitals or areas where penicillin is widely used. Rountree, et al.[47] reports that the number of resistant strains isolated from outpatients is roughly one-half that from in-patients. The present information does not indicate that naturally occurring staphylococci carried by the population at large are frequently resistant to penicillin. Barber,[48] studying the same population group in the same hospital in 1946, 1947, and 1949, reports the incidence of penicillin resistant strains to be 14 per cent, 38 per cent, and 59 per cent in successive years. Howe[49] in 1953 found that 50 to 75 per cent of staphylococci were resistant to penicillin.

The second area in which clinical medicine must deal with bacterial resistance is the group of enterococcal streptococci. Finland[16] points out that some decrease in the efficacy of penicillin is responsible for the present need for large dosages in the treatment of subacute bacterial endocarditis. He states that the widespread use of the antibiotic has resulted in general (not marked) reduction in the sensitivity of strains of the viridans group of streptococci. Dowling[50] reports that in 1951 about one per cent of the cases of subacute bacterial endocarditis have infecting organisms which are highly resistant to penicillin.

Penicillin resistant strains of pneumococci,[51] beta hemolytic streptococci,[52] and gonococci[53] have been produced in the experimental laboratory *in vitro.* This offers interesting speculative material for the future chemotherapy of diseases caused by these micro-organisms.

Cross resistance to antibiotics: Antibiotics with presumably similar mechanisms of action may cause the emergence of forms resistant to each other. This work needs to be explored at greater length for more conclusive information. The present concept of antibiotic cross resistance is given in table I.

TABLE II: ANTIBIOTICS WHICH EXHIBIT CROSS RESISTANCE[50]

Group I	Streptomycin Dihydrostreptomycin Neomycin Streptothricin
Group II	Chloramphenicol Chlorotetracycline Oxytetracycline Tetracycline Penicillin (probably)
Group III	Erythromycin Carbomycin

It should be pointed out that only by promptly terminating infections can the emergence of resistant strains be prevented. This, of course, implies recognition of the etiologic agent and prompt institution of treatment with the drug of greatest efficacy in adequate dosage.

Bacterial resistance has important epidemiological aspects. The widespread use of penicillin among the hosts has led by a selective process to a widespread distribution of staphylococcic strains resistant to penicillin. Since many, though not all, resistant strains of bacteria appear to be as virulent as their sensitive parents, they persist in the population.

Antibiotic combinations: Is is not within the confines of this paper to discuss the problem of combined antibiotic therapy. However, because of its importance and relevance, this large and interesting subject should be mentioned. Indeed, adverse effects of penicillin therapy may be brought about, whether it be through the production of resistant bacteria, failure of treatment, or masking a diagnosis, by combinations of other antibiotics with penicillin.

Jawetz[54] has been engaged in experiments on this

QUESTION?

Have you enrolled in the KANSAS MEDICAL SOCIETY Approved DISABILITY INCOME PLAN?

- **Endorsed and Supervised by Your Society**
- **Non-Cancellable Features**
- **Lifetime Accident Benefits**
- **Five-year Sickness Benefits**
- **30% to 50% Saving in Cost**
- **Over 65% of Eligible Membership Now Enrolled**

Officially approved and endorsed by the Council and the House of Delegates.

For Complete Information Write—Kansas Medical Society, 315 West Fourth, Topeka, Kansas

or

FIRST INSURANCE AGENCY
Washington National Insurance Company
639 New England Building Topeka, Kansas

UNIVERSITY OF KANSAS SCHOOL OF MEDICINE

POSTGRADUATE MEDICAL COURSE

OBSTETRICS

November 8, 9 and 10, 1954

Guest Instructors:

JOHN I. BREWER, M.D., Professor of Obstetrics & Gynecology, Northwestern University Medical School, Chicago, Ill.

F. BAYARD CARTER, M.D., Professor of Obstetrics & Gynecology and Head of Department, Duke University School of Medicine, Durham, N. C.

CARL P. HUBER, M.D., Professor of Obstetrics & Gynecology and Chairman of Department, Indiana University School of Medicine, Indianapolis.

ROBERT A. KIMBROUGH, JR., M.D., Professor of Obstetrics & Gynecology and Chairman of Department, University of Pennsylvania Postgraduate School of Medicine, Philadelphia.

CURTIS J. LUND, M.D., Professor of Obstetrics & Gynecology and Chief of Department, University of Rochester School of Medicine, Rochester, N. Y.

WILLIAM F. MENGERT, M.D., Professor of Obstetrics & Gynecology and Chairman of Department, Southwestern Medical School, University of Texas, Dallas.

Subjects to Be Discussed:

Fibroids and Pregnancy

FIBROIDS AND STERILITY: Do Fibroids Produce Infertility; When and How; Place of Myomectomy in Treating Infertility.
FIBROIDS AND ABORTION: Can One Predict That an Abortion Will Result; After One (Two or Three) Abortion, What Do We Do?
COURSE OF PREGNANCY: The Large Fibroid; Multiple Subserous Fibroids; The "Large" Uterus at Two or Three Months.
ACUTE DEGENERATION: Medical or Surgical; Timing of Operation; Prognosis for Continuation of Pregnancy.
USUAL TYPES OF FIBROIDS: (Obstructing and Non-Obstructing)—The Pediculated Fibroid in Vagina; The Pediculated Fibroid in Cul-de-sac.
COURSE OF LABOR: First Stage; Third Stage.
THE PUERPERIUM AND THE FUTURE: Degree of Expected Involution; When Myomectomy; When Hysterectomy?

Placenta Previa

ETIOLOGY: Is There an Etiology or Is It Merely a Matter of Chance?
DIFFERENTIAL DIAGNOSIS, INCLUDING DEGREE OF PREVIA.
MEDICAL TREATMENT.
SURGICAL TREATMENT.
THIRD STAGE AND NEXT FEW HOURS.
PROGNOSIS FOR CHILD AND FOR NEXT PREGNANCY.

Premature Separation

ETIOLOGY: Role of Toxemia; Venous Pressure.
ROLE OF MEDICAL TREATMENT: Minor Degrees of Separation; Twin Pregnancy; Major Separation.
SURGICAL TREATMENT: When? What?
PROGNOSIS: Baby Now Alive; Future Pregnancies.

The Official Meeting of the Kansas State Obstetrical Society

subject, the results of which have aroused a great deal of interest. Table III is a brief consideration of his results.

TABLE III: ACTION OF ANTIBIOTICS IN COMBINATION

Group I	Penicillin, streptomycin, bacitracin, neomycin (probably polymixin)
Group II	Chloramphenicol, chlorotetracycline, oxytetracycline, erythromycin, carbomycin (sulfonamides behave like group II)

Members of group I are frequently synergistic, occasionally indifferent but never antagonistic to each other.

Members of group II are neither synergistic nor antagonistic with each other but simple additive effects are often observed.

The addition of group I to group II may result in synergism or antagonism, depending in part on the sensitivity of the bacteria to the group I drug.

The material available at the present time has been aptly summarized by the Cornell Conference on Therapy.[55] "There is, as yet, insufficient experience with these matters in human infections to define the meaning of these interesting experiments in relation to therapy. The situation in actual treatment is sufficiently different and unpredictable to make it impossible to put the matter of combined therapy on a sound base, but enough is indicated by these experimental observations to suggest the possibility of doing good as well as harm in the treatment of infections."

Superinfections: This group of adverse effects of penicillin therapy in many aspects is similar to those resulting from penicillin resistance; however, they are sufficiently different to merit separate consideration. The spontaneous occurrence of new infections during the course of treatment with penicillin is a complex and not uncommon problem.

Changes in the bacterial flora of the body during penicillin therapy have been studied by many workers since the drug first became available. As one would expect, the bacteria sensitive to the drug are suppressed by treatment.[56, 57] Weinstein[58] offers two explanations for spontaneous occurrence of new infections during treatment: (1) A suppression of certain groups of bacteria with an increase in other groups usually results in no untoward effects; however, in other cases, such alterations in flora result in a rapid growth of virulent bacteria originally present and not susceptible to the antibiotic. If general resistance to infection in the host be depressed, new infection results, invasion occurs, and new disease develops. (2) Spontaneous occurrence of new infection may also occur as a result of contamination in the administration of penicillin.

Sommer and Favour[59] stress the importance of previous chronic pulmonary disease, of general debility and of the presence of a mixed gram-positive and gram-negative flora in culture of the respiratory tract before treatment in the pathogenesis of infectious complications.

Hypersensitivity studies correlating previous fungus infections and penicillin reactions has been stated. Schnurman[60] described latent tricophyton infections activated by the use of penicillin in which there was actual recurrence of apparently cured infection. Sanchey-Cuenca[61] proposes an interesting possibility in which penicillia may be pathogenic. Penicillia are frequently recovered from the feces in routine stool examinations, being present in the intestine as a saprophyte. The mold may be present in pathogenic form in lung abscesses. Is this a superinfection? It is possible that the presence of penicillia in the host may predispose to sensitization to penicillin. More study is needed before this condition, termed penicilliosis, can be fully understood.

Overgrowth by monilia has been frequently seen with antibiotic therapy. It is less often encountered when penicillin is used alone than when used in combinations, especially with the broad spectrum antibiotics. It is often noted following the local use of penicillin troches or lozenges. Multiple vitamin B preparations have helped clear up these monilial overgrowths.

Signs or symptoms arising during treatment with antibiotics that are not explainable by the existing disease process should be carefully studied. The simple gram-stained smear of pathological material may permit an immediate diagnosis and prompt institution of proper drug therapy. Cultures and sensitivity studies should be done *in vitro* for further advantages in future management.

EFFECTS RELATED TO PENICILLIN AS A PRIMARY TOXIC AGENT

For practical purposes the possibilities of penicillin as a direct toxic agent are negligible except following its intrathecal use. The large number of individuals receiving the drug in massive dosages for the treatment of bacterial endocarditis and syphilis attest to the drug's low toxicity.

The effects of penicillin on the central nervous system are those of stimulation, twitching, and convulsions. The convulsive factor appears to be related to the antibiotic properties of penicillin because seizures diminish directly proportional to the destruction of the antibiotic properties by aging, boiling and acidifying and by dissolving penicillin in alcohol.[62]

Intrathecal reactions include headache, vomiting, vascular collapse, cyanosis, convulsions, unconsciousness, flaccid paralysis of the extremities, with increased spinal fluid cell count and protein values.[63]

Toxic psychosis occurring following intramuscular injection with oral administration is reported but was felt to be due to edematous lesions of the brain resulting from a localized penicillin sensitivity.[62]

From our present knowledge, these effects on the central nervous system are apparently due to irritative

Medical and Surgical Supplies
for Doctors of Medicine
and Hospitals

Munns Medical Supply Co.
512 Kansas Avenue
Topeka, Kansas

MUTUAL TRUST

Offered by

MUTUAL DISTRIBUTORS, Inc.
1016 Baltimore • Kansas City 5, Mo.

Principal Underwriter
and Distributor

PROSPECTUS ON REQUEST

PLEASE SEND ME PROSPECTUS

*Name*_____

*Address*_____

*City*_____ *State*_____

Clarence W. Glassen, State Representative

ANNOUNCING THE TWENTY-FOURTH ANNUAL CONFERENCE
of the
OKLAHOMA CITY CLINICAL SOCIETY
October 25, 26, 27, 28, 1954

DISTINGUISHED GUEST LECTURERS

WALTER B. MARTIN, M.D., *President*, AMERICAN MEDICAL ASSOCIATION, Norfolk, Virginia

GEORGE C. ADIE, M.D., *Surgery*, Director of Surgery, New Rochelle Hospital, New Rochelle, N. Y.

HERBERT S. ALDEN, M.D., *Dermatology*, Assistant Professor of Medicine (Dermatology), Emory University School of Medicine, Atlanta, Georgia.

RUSSELL J. BLATTNER, M.D., *Pediatrics*, Professor of Pediatrics and Chairman of the Department, Baylor University College of Medicine, Houston, Texas.

WILLIAM J. DIECKMANN, M.D., *Obstetrics and Gynecology*, Mary Campau Ryerson Professor and Chairman of the Department of Obstetrics and Gynecology, University of Chicago, Chicago, Illinois.

L. HENRY GARIAND, M.D., *Radiology*, Clinical Professor of Radiology, Stanford University School of Medicine, San Francisco, California.

ARNOLD S. JACKSON, M.D., *Surgery*, Chief-of-Staff, Methodist Hospital, Madison, Wisconsin.

WILLIAM O. JOHNSON, M.D., *Obstetrics and Gynecology*, Professor and Chairman of the Combined Departments of Obstetrics and Gynecology, University of Louisville School of Medicine, Louisville, Kentucky.

LLOYD G. LEWIS, M.D., *Urology*, Professor Clinical Urology, Georgetown University School of Medicine, Washington, D. C.

WILLIAM S. MIDDLETON, M.D., *Internal Medicine*, Dean, University of Wisconsin Medical School, Madison, Wisconsin.

JOHN J. MODLIN, M.D., *Surgery*, Clinical Associate in Surgery, University of Missouri School of Medicine, Columbia, Mo.

JAMES L. POPPEN, M.D., *Neuro-Surgery*, Neurosurgeon to the Lahey Clinic, New England Deaconess, New England Baptist and Boston Psychopathic Hospital, Boston, Massachusetts.

WILLIAM G. SAUER, M.D., *Internal Medicine*, Assistant Professor, Graduate School of Medicine, University of Minnesota, Consultant in Medicine, Mayo Clinic, Rochester, Minnesota.

K. M. SIMONTON, M.D., *Otolaryngology*, Associate Professor of Otolaryngology, Mayo Foundation, Graduate School of Medicine, University of Minnesota, Rochester, Minnesota.

DAVID E. SMITH, M.D., *Pathology*, Assistant Professor of Pathology, Washington University School of Medicine, St. Louis, Mo.

RALPH SOTO-HALL, M.D., *Orthopedics*, Assistant Clinical Professor of Orthopedic Surgery, University of California Medical School, San Francisco, California.

JOSEPH F. VOLKER, D.D.S., *Oral Medicine*, Dean, School of Dentistry, University of Alabama, Birmingham, Alabama.

ROUND TABLE LUNCHEONS
CLINICAL PATHOLOGICAL CONFERENCE — SMOKER — DINNER MEETINGS
GENERAL ASSEMBLIES — POSTGRADUATE PANELS — COMMERCIAL EXHIBITS

Registration fee of $20.00 includes all the above features
***For further information, address:* Executive Secretary, 503 Medical Arts Bldg., Oklahoma City.**

phenomena rather than hypersensitivity. Weinstein, et al.,[64] following a study of intrathecal chemotherapy in bacterial meningitis, have concluded that intrathecal administration of penicillin is attended by a negligible risk of producing nervous system damage provided the dose and dilution of the drug is meticulously adjusted. They state that doses in excess of 30,000 units should not be given into the subarachnoid space of adults and the quantity must be reduced proportionately for children.

EFFECTS ON PATIENT CARE

Adverse effects of penicillin therapy in regard to patient care are numerous. First, it should be recognized that all of the aforementioned adverse effects add greatly to the patient's morbidity. Indeed a more serious illness may be the result of the therapy than the original problem for which the patient sought medical care.

A delay in diagnosis occurs probably more often than is realized. The mechanism by which this happens is considered to be a condition in which enough of the drug is given to mask signs or symptoms without producing a cure. It is often found that bacterial identification cannot be accomplished after penicillin is given. The importance of this fact is obvious when one considers the fact that valuable time may be lost before definitive therapy can be instituted.

Little material is to be found in the literature on this particular subject. Therefore, the diseases listed below are taken from my own experience. The list is not considered complete by any means, but should serve as illustrative material.

Diseases in which a delay in diagnosis resulted from indiscriminate use of penicillin: (1) diphtheria, (2) poliomyelitis, (3) Friedlander's pneumonia, (4) tuberculosis, (5) meningitis, and (6) carcinoma of the lung.

Some illnesses require prolonged treatment. A definitive diagnosis rests upon identification of the bacterial cause. This can usually be done only before antibiotics are given. The most common of these diseases are subacute bacterial endocarditis, osteomyelitis, tuberculosis, and brucellosis.

SUMMARY AND CONCLUSION

The adverse effects of penicillin therapy have been classified and discussed as (1) hypersensitivity reactions, (2) effects related to bacterial alterations, (3) direct toxic reactions, and (4) effects on patient care.

In so far as possible, each of the adverse effects commonly encountered has been considered as to incidence, characteristic features, diagnostic aspects, prophylaxis, and treatment.

Although the clinical evaluation of penicillin is still far from complete, it is the intent to give some perspective of the untoward aspects of treatment with this drug.

Penicillin remains a valuable, indispensable drug. It is, however, very probable that with repeated exposure to penicillin through the years, an increasing number of therapeutic difficulties may be encountered.

Penicillin is a specific chemotherapeutic agent having specific indications for its use. The indiscriminant use of this antibiotic is condemned.

BIBLIOGRAPHY

1. Florey, M. E., and Florey, H. W.: General and local administration of penicillin, Lancet 1:387-397, 1943.
2. Keefer, C. S., Blake, F. G., Marshall, E. K., Jr., Lockwood, J. S., and Wood, W. B., Jr.: Penicillin and the treatment of infection, J.A.M.A. 122:1217-1224, 1943.
3. Rhodes, P. S.: Practical aspects of penicillin administration, M. Clin. North Am. 35:23-26, 1951.
4. Long, P. H.: The clinical use of antibiotics, M. Clin. North Am. 34:307-318, 1950.
5. Chow, B., and McKee, C.: Interaction between crystalline penicillin and human plasma protein, Science 101:67, 1945.
6. Morginson, W. J.: The clinical use of penicillin in dermatology, South. M. J. 38:320, 1945.
7. MacInnis, K. B.: Allergic reactions from handling penicillin, Ann. Allergy 5:102-104, 1947.
8. Barker, A. M.: Allergic reactions to penicillin, Lancet 1:177, 1945.
9. Binkley, G. W., and Brackmole, A.: Dermatitis from penicillin, Arch. Derma. & Syph. 50:326, 1944.
10. Templeton, H. J., Lunsford, C. J., and Allington, H. V.: Cutaneous reactions to penicillin, Arch. Derm. & Syph. 56:325, 1947.
11. Marcovicci, E. E.: Thrush as a sequel to penicillin, N. Y. State J. Med. 46:1361, 1946.
12. Kleinfeld, L.: Sequelae following oral and topical use of penicillin, N. Y. State J. Med. 46:915, 1946.
13. Wheatly, D. P.: Rash after penicillin lozenges, Brit. M. J. 2:448, 1945.
14. Mayer, P. S., Mosko, N. M., Schutz, P. J., Osterman, F. A., Steen, L. H., and Baker, L. A.: Penicillin anaphylaxis, J.A.M.A. 151:351-353, 1953.
15. Keefer, C. S.: Antibiotics: yesterday, today and tomorrow, Ann. Int. Med. 33:582, 1950.
16. Finland, M.: Clinical uses of the presently available antibiotics, antibiotics Annual, 1953-54, Encylopedia Incorporated, 1953.
17. Risman, G., and Boger, W. P.: Human skin sensitivity to penicillin G, BT, O, demonstration of cross-sensitization, J. Allergy 21:425, 1950.
18. Cecil, R. L.: Textbook of Medicine, W. B. Saunders & Co., 1951.
19. Kitchen, D. K., Rein, C. R., Thomas, E. W., and Spoor, H. J.: Reaction to penicillin, Am. J. Syph. 35:578, 1951.
20. Boger, W. P., Sherman, W. B., Schiller, I. W., Siegal, S., and Rose, B.: Allergic reactions to penicillin, J. Allergy 24:383, 1953.
21. Hopkins, J. G., and Lawence, H.: Sensitization to penicillin, J. Allergy 18:251, 1947.
22. Huber, T. E.: Exfoliative dermatitis due to penicillin and streptomycin, Mil. Surgeon 105:4, 1949.
23. Lowell, F. C., and Schiller, I. W.: Measurements in changes in vital capacity as a means of detecting pulmonary reactions to inhaled aerosolized allergenic extracts in asthmatic subjects, J. Allergy 19:100, 1948.
24. Peck, S. M., and Siegal, S.: Successful desensitization in penicillin sensitivity, J.A.M.A. 134:1546, 1947.
25. Rich, A. R.: Hypersensitivity in diseases with especial reference to periarteritis nodosa, rheumatic fever, disseminated lupus erythematosis, and rhematoid arthritis, Harvey Lecture 42:106, 1946-47.
26. Walsh, J. R., and Zimmerman, H. J.: Demonstration of the "L.E." phenomenon in patients with penicillin hypersensitivity, J. Hematology 8:65, 1953.
27. Stokes, J. H., et al.: The action of penicillin in late syphilis, J.A.M.A. 126:73, 1944.
28. Riley, K. A.: Serum-sickness from penicillin, A.M.A. Arch. Derm. & Syph. 65:727, 1952.
29. Reyer, W. A.: The study of the pathogenesis and classification of dermatologie penicillin reactions, Ann. Allergy 10:270, 1952.
30. Kline, C. L., and Highsmith, R. S.: Toxic psychosis resulting from penicillin, Ann. Int. Med. 28:1057, 1948.
31. Feinberg, S. M., Feinberg, A. R., and Moran, C. F.: Penicillin anaphylaxis, non-fatal and fatal reactions, J.A.M.A. 152:114, 1953.
32. Starling, A.: Anaphylactic shock following penicillin therapy in bronchial asthma, J. Allergy 24:543, 1953.
33. Pick, J. F., and Patterson, J. F.: Fatal anaphylactic shock due to penicillin, Brit. Med. J. 2:605, 1953.
34. Everett, R.: Anaphylactic reactions from local use of penicillin, J.A.M.A. 146:1314, 1951.

SECLUSION MATERNITY



FAIRMOUNT HOSPITAL

For Unmarried Girls
Est. 1909

Private sanitarium with certified obstetrician in charge. All adoptions arranged through juvenile court. Early entrance advised.

Rates reasonable. In certain cases work given to reduce expenses.

Write for information

MRS. EVA THOMSON
4911 East 27th St.
Kansas City, Mo.

Have YOU learned the advantages of—

"SAFETY-SEAL" and "PARAGON" ILEOSTOMY, URETEROSTOMY, COLOSTOMY Sets?

They assure the highest standards of COMFORT, CLEANLINESS, and SAFETY for your patients.

Unnoticeable even under girdle or corset. 24-hour control. Odorless. Moisture-proof plastic pouch is inexpensive, disposable.

Construction is adaptable to any enterostomy; militates against waste stagnation; prevents leakage; permits complete emptying.

Order from your surgical supply dealer.
For Medical Journal Reprints and literature write to

THOMAS FAZIO LABORATORIES
Surgical Appliance Division
339 Auburn Street Auburndale 66, Massachusetts
Originators of CLINIC DROPPER

THE SOUTHARD SCHOOL

Intensive individual psychotherapy in a residential school, for children of elementary school age with emotional and behavior problems.

THE MENNINGER CHILDREN'S CLINIC

Outpatient psychiatric and neurologic evaluation and consultation for infants and children to eighteen years.

Department of Child Psychiatry

THE MENNINGER FOUNDATION

J. Cotter Hirschberg, M.D., Director — Topeka, Kansas; Telephone 3-6494

Deformity Appliances of Quality

Orthopedic and Surgical Appliances

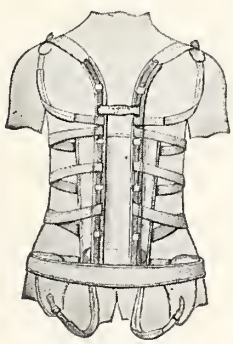
Artificial Limbs
Trusses

Abdominal Supports

Elastic Hosiery

Foot Supports

Surgical Corsets



Taylor Back Brace
Made to Order in
Our Own Factory

P. W. HANICKE MFG. CO.
1009 McGee St. Victor 4750
KANSAS CITY, MO.

THE
MEDICAL PROTECTIVE
COMPANY
FORT WAYNE, INDIANA

PROFESSIONAL PROTECTION
EXCLUSIVELY
SINCE 1899

specialized service
assures *"know-how"*

TOPEKA Office:
J. E. McCurdy, Rep.,
1035 Randolph Avenue,
Telephone 2-3027

35. Leibowitz, H., and Schwartz, E.: An unusual reaction to penicillin, Ann. Allergy 8:688, 1950.
36. Editorial: Fatal allergic reactions to penicillin, J. Allergy 23:383, 1952.
37. Blanton, W. B., and Blanton, F. M.: Unusual hypersensitiveness, J. Allergy 24:405, 1953.
38. Thomson, W. O.: Sudden death following an injection of penicillin, Brit. M. J. 2:70, 1952.
39. Seigal, S., Steinhardt, R. W., and Gerber, R.: Fatal and near fatal penicillin anaphylaxis, J. Allergy 24:1, 1953.
40. Editorial: Penicillin an allergic hazard, U. S. Armed Forces M. J. 1:1155, 1950.
41. Waldbott, G. L.: Anaphylactic death from penicillin, J.A.M.A. 139:526, 1949.
42. Abraham, E. P.: Further observation on penicillin, Lancet 2:177, 1941.
43. Prissick, F. H.: Antibiotic-resistant staphylococci and related infections, Am. J. Med. Sc. 225:299, 1953.
44. Klimek, J. W., Cavallito, C. J., and Bailey, J. H.: Induced resistance of Staphylococcus aureus to various antibiotics, J. Bacti. 55:139, 1948.
45. Barber, K.: Coagulase-positive staphylococci resistant to penicillin, J. Path. & Bacti. 59:373, 1947.
46. Fairlie, C. W., and Kendall, R. E.: Fatal Staphylococcus enteritis following penicillin and streptomycin therapy, J.A.M.A. 153:90, 1953.
47. Rountree, P. M., Barbour, R. G. H., and Thompson, E. I.: Incidence of penicillin resistant and streptomycin resistant staphylococci in a hospital, Lancet 1:435, 1951.
48. Barber, M., and Roywadowska-Dowzenke, M.: Infection by penicillin resistant staphylococci, Lancet 2:641, 1948.
49. Howe, C. W.: The treatment of staphylococcal infections, Med. Clin. N. Am., Sept. 1461, 1953.
50. Dowling, H. F.: The effect of the emergence of resistant strains on the future of antibiotic therapy, Antibiotics Annual 1953-54, Medical Encylopedia Incorporated, 1953.
51. Rake, G., McKee, C. M., Hamre, D. M., and Houck, C. L.: Studies on penicillin, part II: Observation on therapeutic activity and toxicity, J. Immunol. 48:271, 1944.
52. Gezon, H. M.: Penicillin resistance acquired by Group A organisms, Proc. Soc. Exper. Biol. Med. 67:208, 1948.
53. Bohn, J. M., Ackerman, H., and Carpenter, C. M.: Development *in vitro* of penicillin resistant strains of the gonococcus, Proc. Soc. Exper. Biol. Med. 58:21, 1945.
54. Jawetz, E.: Combined antibiotic action, Antibiotics Annual, 1953-54, Medical Encylopedia Incorporated, 1953.
55. Cornell Conference on Therapy: Therapeutic use of combinations of antibiotic agents, N. Y. State Med. J. 53:1228, 1953.
56. Lipman, M. O., Coss, J. A., and Boots, R. H.: Changes in the bacterial flora of the throat and intestinal tract during prolonged administration of penicillin, Am. J. Med. 4:702, 1948.
57. Long, D. A.: Effect of penicillin on bacterial flora of the mouth, Brit. Med. J. 2:819, 1947.
58. Weinstein, L.: The spontaneous occurrence of new bacterial infections during the course of treatment with streptomycin or penicillin, Am. J. Med. Sci. 214:56, 1947.
59. Sommer, L. S., and Favour, C. B.: Biologic complications of penicillin therapy, Am. J. Med. 7:511, 1949.
60. Schnurman, A. G.: Five cases of latent trycophyton infection activated by the use of penicillin, Virginia Med. Monthly 73:281, 1946.
61. Sanchez-Cuenca, B.: Penicilliosis and hypersensitiveness to penicillin, J. Allergy 21:176, 1950.
62. Walker, A. E., and Johnson, H. C.: Convulsive factor in commercial penicillin, Arch. Surg. 50:69, 1949.
63. Morginson, W. J.: Toxic reactions accompanying penicillin therapy, J.A.M.A. 132:915, 1946.
64. Weinstein, L., Goldfield, M., and Adams, D.: The study of intrathecal chemotherapy in bacterial meningitis, Med. Clin. N. Am. Sept. 1363, 1953.

Nationally advertised Surgical Supplies and Equipment have been placed at Topeka, Joplin, Kansas City and St. Joseph for your convenience by—

GOETZE NIEMER COMPANY

Management by Dr. W. F. Goetze, member of the American Medical Ass'n, assures intelligent servicing of your orders.

It's Convenient to Buy Kanox

THERAPEUTIC OXYGEN AND ANESTHETIC GASES



Oxygen Therapy
Regulators and Equipment
Repair Service for
Regulators, Flowmeters
and Humidifiers

Nitrous Oxide
Ethylene
Carbon Dioxide
Helium
Oxygen
Cyclopropane

KANSAS OXYGEN, Inc.

Phone 5-5551 HUTCHINSON, KANSAS Box 551

PITOCIN®

AN OXYTOCIC OF CHOICE

PITOCIN is widely used in obstetrics because of its physiologic effect on uterine musculature. In addition, the fact that it is notably free from vasopressor action is often a significant advantage. Intravenous administration of diluted **PITOCIN** in emergencies makes possible ready control of dosage and response.

PITOCIN is valuable in treatment for primary and for secondary uterine inertia, for postpartum hemorrhage due to uterine atony, for the third stage of labor, for induction of labor, and during cesarean section to facilitate suturing the uterine wall.

*Kaufman, R. H.; Mendelowitz, S. M., & Ratzan, W. J.: *Am. J. Obst. & Gynec.* 65:269, 1953.

PITOCIN (oxytocin injection, Parke-Davis) is supplied in 0.5-cc. (5-unit) ampoules, and in 1-cc. (10-unit) ampoules, in boxes of 6, 25, and 100. Each cc. contains 10 international oxytocic units (U.S.P. units).

MEDICAMENTA VERA

Parke, Davis & Company

TABLE OF CONTENTS

OCTOBER, 1954

ORIGINAL ARTICLES

The Value of Gastroscopy—Henry Laurens, M. D., Salina, Kansas 559

The Management of Traumatic Rupture of the Left Superior Pulmonary Vein—Harry R. Custer, M.D., Colby, Kansas, and Karl P. Klassen, M.D., Columbus, Ohio 563

Paroxysmal Ventricular Tachycardia Occurring During a Two-Step Exercise Test—Robert A. Jordan, M.D., Kansas City, Kansas . . 566

Current Trends in Treatment of Congenital Megacolon—William N. Harsha, M.D., Oklahoma City, Oklahoma 569

EDITORIALS

Maternal Mortality Study 577

Federal Aid in Kansas 577

United Funds 578

MISCELLANEOUS

President's Page 576

Recent Trends in Therapy of Carcinoma of the Breast—Tumor Conference 580

The Medical Management of Peripheral Arteriosclerosis—John D. King, M.D.—Senior Thesis 592

Entered as second-class matter at the post office at Fulton, Missouri, June 19, 1953, under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of October 3, 1917, authorized on July 2, 1918. Office of Publication: 1201-05 Bluff Street, Fulton, Missouri.

THE JOURNAL OF THE KANSAS MEDICAL SOCIETY

Editorial Board

ORVILLE R. CLARK, M.D., *Editor,* Topeka

JOHN W. CAVANAUGH, M.D., Topeka

DAVID E. GRAY, M.D., Topeka

RICHARD GREER, M.D., Topeka

DWIGHT LAWSON, M.D., Topeka

Associate Editors

DONALD P. TREES, M.D., Wichita

GLEN R. SHEPHERD, M.D., Kansas City

Information

Ownership: The Journal is a non-profit publication owned and published monthly by the Kansas Medical Society.

Subscription: A year's subscription to the Journal is included in membership in the Kansas Medical Society, with $2.00 of each member's dues apportioned to the Journal. Rates to others, except in foreign countries, $4.00 per year or 60c per copy.

Material: Scientific articles, editorials, and data of general interest are invited from all members. Articles are to be submitted on condition that they are contributed solely to this publication. A right is reserved to reject any material deemed unsatisfactory.

Manuscripts: Only manuscripts that are typewritten on one side, double spaced, and original copies can be accepted. Manuscripts will be returned upon request.

Advertising: All advertising contracts, and all copy from advertisers under contract are subject to approval of the editorial board. Copy should be received by the 20th of the month immediately preceding the month of publication.

Non-Responsibility: Although an effort is made to publish only accurate articles and legitimate advertisements, the Journal denies legal responsibility for any statements, opinions, or advertisements appearing under the names of contributors or concerns.

PAULINE FARRELL, Managing Editor
and Advertising Manager

OLIVER E. EBEL, Business Manager

THE KANSAS MEDICAL SOCIETY

President.....MURRAY C. EDDY, M.D., Hays

Past President.....LUCIEN R. PYLE, M.D., Topeka

President-Elect.....JOHN M. PORTER, M.D., Concordia

First Vice-President...CLYDE W. MILLER, M.D., Wichita

Second Vice-President, CONRAD M. BARNES, M.D., Seneca

Constitutional Secretary, JAMES A. BUTIN, M.D., Chanute

Treasurer.....JOHN L. LATTIMORE, M.D., Topeka

A.M.A. Delegate, 1954-1955, GEORGE F. GSELL, M.D., Wichita

A.M.A. Delegate, 1955-1956, LAURENCE S. NELSON, M.D., Salina

Councilors and Councilor Districts

First District, FREDERICK E. WRIGHTMAN, M.D., Sabetha

Second District...GLENN R. PETERS, M.D., Kansas City

Third District.....H. PENFIELD JONES, M.D., Lawrence

Fourth District....CHARLES E. VESTLE, M.D., Humboldt

Fifth District, SEVERT A. ANDERSON, M.D., Clay Center

Sixth District.....FLOYD C. TAGGART, M.D., Topeka

Seventh District.....TO BE ANNOUNCED

Eighth District.....JAMES E. HILL, M.D., Arkansas City

Ninth District....LAURENCE S. NELSON, JR., M.D., Salina

Tenth District.....HAROLD M. GLOVER, M.D., Newton

Eleventh District....NORTON L. FRANCIS, M.D., Wichita

Twelfth District.....CYRIL V. BLACK, M.D., Pratt

Thirteenth District..LLOYD W. REYNOLDS, M.D., Hays

Fourteenth District....JUSTIN A. BLOUNT, M.D., Larned

Fifteenth District....ROBERT G. KLEIN, M.D., Dodge City

Sixteenth District.....JAMES L. JENSON, M.D., Colby

Seventeenth District.....H. PRESTON PALMER, M.D., Scott City

OLIVER E. EBEL, EXECUTIVE SECRETARY
RUEBEN M. DALBEC, EXECUTIVE ASSISTANT

Executive Office: 315 West Fourth Street, Topeka

THE JOURNAL
of the
KANSAS MEDICAL SOCIETY

Copyright, 1954, by The Kansas Medical Society

Volume LV OCTOBER, 1954 No. 10

The Value of Gastroscopy

Henry Laurens, Jr., M.D.
Salina, Kansas

The first attempts at gastroscopy were made in 1868 by Kussmaul in Europe after he watched a sword swallower at a fair. He reasoned that a tube with a lamp in it could be swallowed as easily as a sword, but difficulty with illumination caused the method to be abandoned. Then, in 1881, Mikulicz introduced his gastroscope which overcame some of the technical problems, but, as they seemed insurmountable, he finally discontinued his work. From 1881 until the early 1920's, numerous flexible instruments were tried, but since these proved to be impracticable, the principle of rigid, straight, optical gastroscopes prevailed.

In the 10-year period from 1922 to 1932, Dr. Rudolf Schindler constructed a new flexible gastroscope and developed the endoscopic pathology of the stomach. In 1934, Dr. Schindler was invited to the University of Chicago as a visiting professor in order to introduce gastroscopy to this country. Since that time the modern flexible gastroscope has become so popular that it is used in most clinics and hospitals. Through the efforts of the American Gastroscopic Society many new aspects of gastroscopy have been introduced. With the development of the operating gastroscope by Benedict more than five years ago, we now have the diagnosis of gastric disease on a level with that of any other organ which is accessible to endoscopic methods.

The indications for gastroscopy are as follows:

1. Some chronic ulcers
2. Ulcer syndrome with no diagnosis
3. Follow healing of benign ulcer
4. Pre-operative check
5. Differential diagnosis between chronic gastritis and gastric neurosis
6. Differential diagnosis between gastric carcinoma and benign ulcer
7. Gross gastric hemorrhage
8. Questionable narrowed antrum
9. Postoperative stomach
10. Atrophic gastritis
11. Pernicious anemia
12. Any unexplained suspected stomach disease

Some small gastric ulcers cannot be seen by the roentgenologist and are diagnosed only by gastroscopy. Many cases of suspected ulcer with negative x-ray findings are seen, and direct visualization of the gastric mucosa will often show some form of gastritis. While we all are well aware of the dangers and tremendous responsibility we assume when we decide to treat a gastric ulcer medically, there are certainly some lesions, especially those seen in younger individuals, which are typically benign and which can be followed by both x-ray and gastroscopy. To be able to directly observe an ulcer heal and leave perfectly normal mucosa certainly aids the clinician and roentgenologist in their close observation of a gastric lesion.

Gross gastric hemorrhage is a definite indication for gastroscopy, and occasionally the diagnosis can safely be made when early x-ray examination seems dangerous. In the series of cases which will be presented later, there are two examples of gastric bleeding which were not visualized by x-ray, one a bleeding polyp[1] and the other an acute superficial erosion. Hypertrophic and superficial gastritis will occasionally produce massive hemorrhage.

Presented at the regional meeting of the American College of Physicians, Topeka, Kansas, March 19, 1954.

The x-ray diagnosis of gastrojejunal ulcer is often difficult in the postoperative stomach, especially in a patient who has had only a gastrojejunostomy and who presents the differential diagnosis between a recurring duodenal ulcer and a stomal ulcer. Most gastrojejunal lesions can be seen easily by the gastroscopist.

Atrophic gastritis and the gastric atrophy which accompanies pernicious anemia are both considered to be precancerous lesions. Several investigators have reported the incidence of carcinoma developing in patients with pernicious anemia to be as high as 10 per cent. Most polyps develop in stomachs with achlorhydria and atrophic changes. Therefore, any patient who presents these changes on gastroscopy should have the benefit of serial examinations every 6 to 12 months.

The contraindications for gastroscopy are divided by Schindler[2] into absolute and relative contraindications.

The absolute contraindications are as follows:

1. Non-cooperation of the patient
2. Obstruction of esophagus or cardia
3. Aneurysm of descending aorta
4. Acute corrosive and acute phlegmonous gastritis

The relative contraindications are as follows:

1. Acute and chronic tonsillitis and pharyngitis
2. Cardiac insufficiency
3. Dyspnea
4. Cardiospasm
5. Pulsion diverticulum of esophagus
6. Esophageal varices
7. Kyphosis and scoliosis of thoracic spine
8. Psychosis
9. Hiatal hernia

As indicated, these are only relative contraindications, and careful handling of the cardiac patient will usually lead to successful gastroscopy. The flexible scope can often be passed in spite of a moderate thoracic kyphosis, and an hiatal hernia usually does not prevent instrumentation.

The following is a discussion of approximately 250 gastroscopic observations with a detailed presentation of 200 successful gastroscopies. There were seven unsuccessful examinations which were due to the following causes:

1. Lack of cooperation	2
2. Lack of cooperation and marked kyphosis	1
3. Lack of cooperation due to language barrier	1
4. Hiatal hernia	3

The next table enumerates the different conditions visualized in the 200 successful observations.

1. Normal stomach	81
2. Hypertrophic gastritis	39
3. Atrophic gastritis or gastric atrophy	29
4. Carcinoma of stomach	12
5. Benign gastric ulcer	12
6. *Benign tumors:*	
Bleeding polyp	1
Prolapsing polyp	1
7. Superficial gastritis	5
8. *Postoperative stomach:*	
Normal gastroenterostomy	7
Giant hypertrophic gastritis	1
Superficial gastritis	2
Stomal ulcer	1
Recurrent carcinoma	1
9. *Miscellaneous conditions:*	
Gastric atrophy associated with pernicious anemia	2
Superficial erosion	1
Tumor simulating gastritis	1
Prolapse of gastric mucosa	1
Gastrostomy	1
Benign pyloric obstruction	1
Phytobezoar	1

The gastroscopic diagnosis in eight cases proved to be wrong either by subsequent surgery or by clinical follow-up. Seven of these have been listed in a previous discussion.[14] One additional case of benign ulcer was incorrectly diagnosed as infiltrating carcinoma.

The following table is a correlation of x-ray with gastroscopic diagnoses:

X-ray Diagnosis	*Gastroscopic Diagnosis*
Possible lesion lesser curvature	Carcinoma
Normal gastroenterostomy	Mal-functioning stoma
Diffuse polyposis	Normal
Carcinoma of pylorus	Normal
Normal	Bleeding erosion
Carcinoma of stomach	Phytobezoar
Normal	Gastric ulcer
Carcinoma	Tumor simulating gastritis
Normal	Stomal ulcer
Normal	Bleeding polyp
Prolapsing antral polyp	Normal
Normal	Hypertrophic gastritis 39
Normal	Atrophic gastritis or gastric atrophy 29

Therefore, there were 11 cases in which the correct diagnosis was made only by gastroscopy. The group of 39 cases of hypertrophic and of 29 cases of atrophic gastritis or gastric atrophy is included only to suggest that the gastroscopic findings may possibly help the clinician in treating certain patients with symptoms but negative x-rays. It is not intimated that the radiologist should have made the correct diagnosis in this group of patients.

Recently Palmer[3] has shown that there is probably no true correlation between the gastroscopic appearance and the pathological findings in hypertrophic gastritis, for biopsies of the typical cobblestone lesions are often normal. He suggests that there may be an abnormality involving the muscularis mucosae and that the symptoms and gastroscopic findings are

related to minute contractions therein. Much more study of this important subject will be necessary.

The relationship between x-ray and gastroscopy is a very important one, and comparisons of cases of gastric carcinoma have revealed that both procedures are necessary in order to increase our percentage of accuracy of diagnosis of such lesions. Large series of cases of both gastric cancer and ulcer have been analyzed by Palmer,[4] and results have shown that in carcinoma, gastroscopy established a correct diagnosis in about 77 per cent and x-ray in about 84 per cent. Certainly, when the two are used together, this percentage goes up.

Moersch and Kirklin,[5] in an analysis of 100 cases of cancer of the stomach at the Mayo Clinic, found the initial gastroscopic impression to be correct in 64, indefinite in 13, and incorrect in 23 cases; and they have shown that close cooperation between the roentgenologist and gastroscopist is of great importance in improving the chances of earlier diagnosis of carcinoma of the stomach.

Hardt,[6] in a recent analysis of 203 cases of gastric ulcer, found that individually x-ray gave a correct diagnosis in only 22.2 per cent of the malignant and 35.7 per cent of the benign lesions, and gastroscopy in 44.5 per cent of the malignant and 39.3 per cent of the benign lesions. However, when x-ray and gastroscopy were combined and repeated after short intervals of treatment, the two methods supplemented each other and gave a higher index of accuracy. He believes that no patient with gastric symptoms should be considered as having an adequate check-up without gastroscopy.

Schindler,[7] on the other hand, in a summary of 273 gastric ulcers with an adequate follow-up, found an incorrect diagnosis in only 20 cases, or 7.3 per cent. He believes that the percentage of correct gastroscopic diagnoses is high and that it is probably more valuable than any other method in the correct differential diagnosis of gastric ulcer.

Baker et al.,[8] in an investigation of the diagnostic accuracy of histologically proved lesions observed over a five-year period, concluded that most gastroscopic errors were due to inadequate visualization of the lesion and that x-ray study was less accurate than gastroscopy when the lesion could be seen. Their experience was similar to that of others in that the combined accuracy of the two methods in the diagnosis of gastric cancer was 91.5 per cent, while that of x-ray and gastroscopy individually was only 68 per cent and 73 per cent.

Deutsch[9] believes that the patient with a gastric lesion can be observed more adequately by gastroscopy than by x-ray so far as the diagnosis, treatment, and prognosis of the disease are concerned.

It is my policy to attempt to gastroscope every patient with unexplained upper abdominal symptoms. When certain precautions are observed, the examination is safe and easy, and from routine questioning of my patients, I have learned that many of them prefer gastroscopy under the proper topical anesthesia and sedation to a gastric analysis.

I would like to stress the point that gastroscopy is often done in the office and that the patient may return home in two to three hours. My routine is to give atropine by hypo about one hour prior to instrumentation. At the beginning of the examination I spray the throat with a solution of about 2 to 3 per cent pyribenzamine. This has been used successfully by Reynolds et al.[10] and avoids the complications of a pontocaine reaction. I obtain satisfactory topical anesthesia and absence of the gag reflex with PBZ,* and have used this preparation entirely for more than one year. Atropine is usually necessary to prevent excess salivation caused by the PBZ.

After five to ten minutes I give the patient from 50 to 100 milligrams of demerol intravenously. With this latter method I am able to insure both marked relaxation and excellent cooperation of the patient. Demerol given intravenously is excreted rapidly, and the patients thus prepared are able to go home in one to two hours after the examination. I have been using this method with great success for approximately one year. The intravenous use of demerol in gastroscopy has recently been highly recommended by Cimoch and Wirts[11] without any spray being used at all.

After the demerol is given, I introduce a No. 12 Ewald tube in order to empty the stomach of any overnight secretions. The gastroscope is then passed with the patient in the left lateral position. After examination in this standard position, as suggested by Berry,[12] the patient is usually rotated onto the back, as better visualization of the entire antrum and pyloric sphincter is thereby possible. Gastroscopy is never really satisfactory unless the entire antrum and sphincter are seen. Following withdrawal of the gastroscope, I introduce a plastic tube containing a wing-brush. This has been described by Ayre,[13] is used to obtain smears for gastric cytology, and is another aid in the early diagnosis of gastric lesions.

The following seven cases will illustrate the value of gastroscopy. Five of these have been discussed previously.[14]

1. A 65-year-old white female complained of vague indigestion for one year. There was a moderate loss of weight, a hypochromic anemia, no free HCl following histamine, and the stool was positive for occult blood. X-rays revealed a suspicious lesion on the lesser curvature above the angle, but repeat

* Kindly furnished by Ciba Pharmaceutical Products, Summit, New Jersey.

studies did not indicate the exact nature of the lesion. Gastroscopy revealed a typical infiltrating carcinoma of the stomach. Subsequently surgery confirmed the gastroscopic diagnosis.

2. A 49-year-old white male had a gastrojejunostomy in 1948 for a chronic duodenal ulcer with pyloric obstruction. In 1951 he had a return of ulcer symptoms with a 20-pound weight loss and occult blood in his stools. X-ray examination revealed a chronic duodenal ulcer with some malfunction and edema of the gastrojejunostomy. However, gastroscopy revealed a definite jejunal ulcer, and subsequent surgery confirmed this with the finding of a large chronic jejunal ulcer.

3. A 30-year-old white female entered the hospital after the sudden onset of tarry stools and indigestion. She had been taking rather heavy doses of salicylates for rheumatism. Red blood count was 4,000,000 with hemoglobin 80 per cent. Free HCl was 36 degrees. X-ray examination of the upper gastrointestinal tract was normal. Gastroscopy, however, revealed a definite small superficial hemorrhagic erosion on the lesser curvature of the antrum just proximal to the pyloric sphincter. Following withdrawal of salicylates, the patient had no further gastrointestinal symptoms.

4. A 47-year-old white male gave a history of vague epigastric pain, indigestion, and weight loss for two years. Blood count was normal, free HCl was 68 degrees, and the stool was negative for occult blood. X-ray examination revealed a large ulcerated mass occupying most of the antrum, and the roentgenologist made the diagnosis of far-advanced carcinoma. The patient was therefore given a bland diet and antispasmodics. However, he returned in nine months feeling much improved. Repeat x-ray examination revealed little change except for more antral constriction. Following this, gastroscopy revealed marked hypertrophic changes with large, nodular, edematous folds, which apparently represented a tumor-simulating hypertrophic gastritis. The patient continued to do well, gained ten pounds in the next seven months, and had complete disappearance of symptoms on medical therapy.

5. A 71-year-old white male had had vague indigestion for 30 years. He had been called neurotic at a large reputable clinic, and recent x-rays elsewhere had been reported negative. X-rays revealed an antral tumor which prolapsed into the duodenum. Gastroscopy revealed a benign polyp which prolapsed back and forth through the pyloric sphincter. Subsequent surgery confirmed the impression that the lesion was benign.

6. A 60-year-old white female entered the hospital several hours after marked hematemesis of bright red blood, soon followed by tarry stools. Past history revealed that she had had four previous similar episodes during the past 12 years, each being treated by bed rest and iron. The patient had refused surgery since no cause for the bleeding had been determined. After admission, the red blood count was 2,950,000 and hemoglobin was 64 per cent. All other laboratory work was normal. Free HCl following histamine was 32 degrees. X-ray examination of the upper gastrointestinal tract two days later revealed only a large diverticulum on the medial side of the second portion of the duodenum. Fluoroscopic examination had been difficult because of the patient's weakened condition and the fact that she weighed 250 pounds. Gastroscopic examination four days after x-ray revealed a large bleeding polyp on the lesser curvature of the antrum just proximal to the pylorus. Subsequent gastrotomy confirmed this, and a 1.5 x 1.0 cm. bleeding benign polyp was removed.

7. A 63-year-old white male gave a history of indigestion for ten years and of a perforated gastric ulcer in 1949. He was next seen in 1950, at which time x-ray revealed a duodenal ulcer. He was not seen again until February, 1953, at which time he had a marked increase in symptoms with weight loss, anemia, and occult blood in his stools. At that time, x-ray reported a large irregular filling defect in the body of the stomach interpreted as a carcinoma. Free HCl after histamine was 75 degrees. Gastroscopy revealed an acute benign gastric ulcer on the anterior wall of the distal body and also a large brownish-black multi-lobulated lesion in the body of the stomach. A bezoar was suspected, and a history of eating persimmons for 15 years was obtained. Repeat gastroscopy ten days later revealed the ulcer to be healed, and a definite diagnosis of a phytobezoar was made. At surgery, one large 4 x 5 x 8 cm. bezoar and two smaller 3 x 4 cm. bezoars were removed from the stomach. The patient has had no further difficulty, and it seems most likely that the bezoar was present in 1949, caused an acute ulcer with perforation, but was unrecognized.

SUMMARY

A brief background of the history of gastroscopy is presented.

Experience with approximately 250 gastroscopic observations is reviewed with a detailed description of 200 successful cases.

The indications and the absolute and relative contraindications for gastroscopy are tabulated.

The diagnosis in eight cases was definitely proved incorrect either by clinical follow-up or by surgery.

There were 11 cases where the correct diagnosis was made only by gastroscopy. A representative group of 39 cases of hypertrophic gastritis and of

29 of atrophic gastritis or gastric atrophy is included only to suggest that the gastroscopic findings may possibly help the clinician in treating these patients with symptoms, but negative x-rays.

The importance of the combined use of x-ray and gastroscopy is stressed.

Routine preparation for gastroscopy is outlined, followed by a presentation of seven representative cases.

Gastroscopy should be considered as a routine method of examination in every case of unexplained upper gastrointestinal symptoms.

BIBLIOGRAPHY

1. Laurens, H., Jr.: Diagnosis of gastric bleeding, Bull. Amer. Gastroscopic Soc. 3:1 (Oct.) 1953.
2. Schindler, R.: Gastroscopy, the endoscopic study of gastric pathology, Univ. of Chicago Press, Second Edition, 1950.
3. Palmer, E. D.: The significance of hypertrophic and of superficial gastritis, Bull. Amer. Gastroscopic Soc. 3:7 (Oct.) 1953.
4. Palmer, E. D.: Stomach disease as diagnosed by gastroscopy, 1949. Lea and Febiger.
5. Moersch, J. J., and Kirklin, B. R.: Gastroscopy and its relation to roentgenology in the diagnosis of carcinoma of the stomach, Gastroenterology 7:285, 1946.
6. Hardt, L. L.; Steigmann, F., and Weiss, A.: Gastric ulcer: its diagnosis and differentiation by gastroscopic and roentgenological examination, Am. J. Dig. Dis. 20:253, 1953.
7. Schindler, R., and Desneux, J.: Gastroscopic diagnosis in 273 gastric ulcers, Gastroenterology 24:328, 1953.
8. Baker, L.; Gorvett, E. A., and Spellberg, A.: Diagnostic accuracy of gastroscopy in neoplasms of stomach, Cancer 5:1, 116, 1952.
9. Deutsch, E.: Gastroscopy. Contributions to the diagnosis of benign and malignant lesions of the stomach. N. Eng. Jour. of Med. 250:468, 1954.
10. Reynolds, J.; Kahn, A. G., Jr., and Levy, J. S.: The use of antihistaminic drugs for local anesthesia for gastroscopy, Gastroenterology 14:535, 1950.
11. Cimoch, P. J., and Wirts, C. W.: Meperidine hydrochloride used intravenously before gastroscopy, Jour. Amer. Med. Asso. 153:1004, 1953.
12. Berry, L. H.: Newer trends and technics in gastroscopy, Rev. Gastroenterol. 18:791, 1951.
13. Ayre, J. E., and Oren, B. G.: A new rapid method for stomach-cancer diagnosis: the gastric brush, Cancer 6:1177, 1953.
14. Laurens, H., Jr., and Northrip, R. U.: The value of gastroscopy, Jour. Okla. State Med. Asso. 47:9, 1954.

The Management of Traumatic Rupture of The Left Superior Pulmonary Vein

Harry R. Custer, M.D., and Karl P. Klassen, M.D.
Colby, Kansas Columbus, Ohio

Traumatic intrathoracic hemorrhage producing shock or cardio-respiratory embarrassment is an acute surgical emergency. The institution of immediate therapy is imperative inasmuch as interference with cardio-respiratory physiology may be rapidly fatal.

Without performing a thoracotomy it is not always possible to determine the site or the extent of intrathoracic bleeding following trauma. No surgeon would hesitate to explore a patient suffering from traumatic intraabdominal hemorrhage, yet many still advocate the mere institution of drainage for traumatic hemothorax. Lethal complications such as profound shock, tension hemothorax, alveolar flooding, and cardiac tamponade may suddenly develop. Thus it is obvious that surgical intervention to control the hemorrhage is apt to be the most conservative therapy.

From the Thoracic Surgery Division of the Department of Surgery at Ohio State University Medical Center, Columbus, Ohio.

The following case of traumatic rupture of the superior pulmonary vein is presented to emphasize the management of this problem and to ascertain the cardio-respiratory function after ligation of a major pulmonary vessel.

CASE REPORT

History: B. J. S., Number 52-96804, a 12-year-old boy, was involved in an auto accident at 3:40 p.m., October 15, 1952. The patient was seen in the emergency room at Children's Hospital in Columbus, Ohio, at 4:00 p.m., suffering from marked traumatic and hemorrhagic shock and exhibiting severe cardio-respiratory distress.

Physical Examination: Blood pressure was 80/50, pulse 140, respiration 46. There was obvious subcutaneous emphysema over the left lateral chest. Over the third, fourth, and fifth left lateral ribs was a 10-centimeter defect in the thoracic cage with

the overlying skin intact. The defect expanded markedly with each expiration and receded with each inspiration. The patient exhibited paradoxical respirations due to this defect. There were no breath sounds audible in the left chest. The heart tones were distant and shifted to the right.

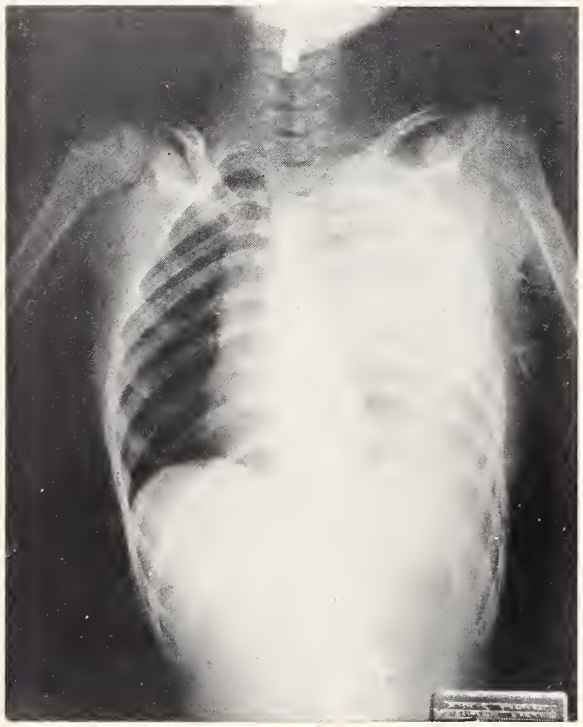


Figure 1

Hospital Course: A pressure dressing was immediately applied over the defect in the thoracic cage. Nasal oxygen and intravenous plasma were started. A needle thoracentesis of the left pleural space revealed blood. An x-ray of the chest showed fluid in the left pleural space producing a compression of the left lung and a marked mediastinal shift to the right. There was a fracture of the left fifth rib anteriorly with downward displacement and overriding of the fragments. Subcutaneous emphysema was present in the left chest wall (Figure 1).

The patient was immediately taken to surgery. Blood transfusions were started and under local anesthesia a thoracotomy tube was introduced into the left pleural space. A large amount of fresh blood under considerable pressure was obtained.

Under general anesthesia a standard left anterolateral submammary thoracotomy was done. The left pleural space contained approximately 1,000 to 1,500 cc. blood. The left lung was collapsed, the mediastinum was shifted to the right, and the lingula exhibited two large lacerations. The left superior pulmonary vein was found to be completely lacerated just distal to the pericardium, and bleeding was profuse.

The pulmonary vein was ligated proximally and distally with 00 silk. After ligation of the vein the upper lobe of the lung rapidly became congested. However, it was deemed unnecessary to perform an upper left lobectomy. During repair of the lacerations of the lingula, the patient developed a hemopericardium and a cardiac tamponade. The pericardium was opened, and the blood was evacuated. The heart exhibited marked irregularity, and one-half per cent procaine was applied to the myocardium. The pericardium was loosely sutured.

Two thoracotomy tubes were placed in the left chest, one draining from the lower postero-lateral chest and the other from the upper antero-lateral chest. The chest was closed in a routine manner, and no attempt at immobilization of the fractured rib was made. Both thoracotomy tubes drained into an under water seal with the lower one connected to a Stedman pump.

On the first postoperative day the patient's temperature reached 103 degrees, and he developed a harsh systolic apical cardiac murmur. An electrocardiogram revealed only a tachycardia of 128. An x-ray of

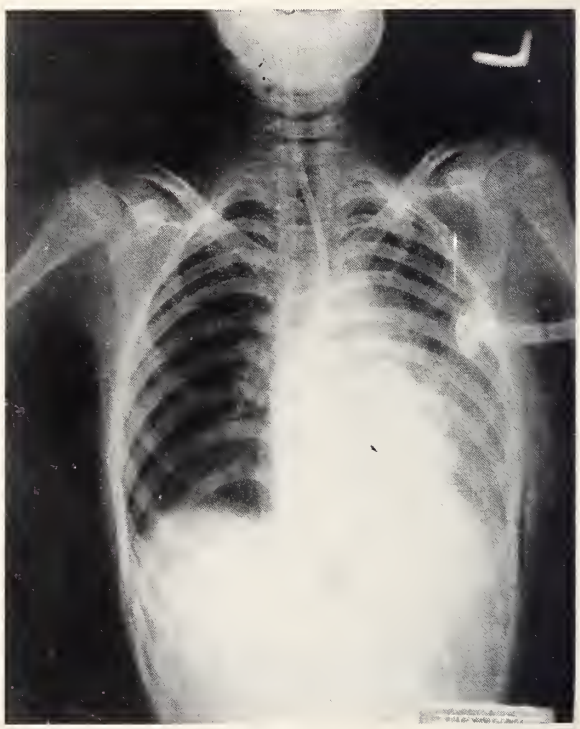


Figure 2

the chest showed little fluid in the left pleural space. The mediastinum was in the midline. The lung was well expanded and there was no abnormality of the cardiac shadow. There was some infiltration at the lower pole of the left hilum (Figure 2).

On the second postoperative day the cardiac murmur disappeared and the x-ray showed an accumulation of fluid in the left chest. Recheck films on the fourth postoperative day showed a regression of the

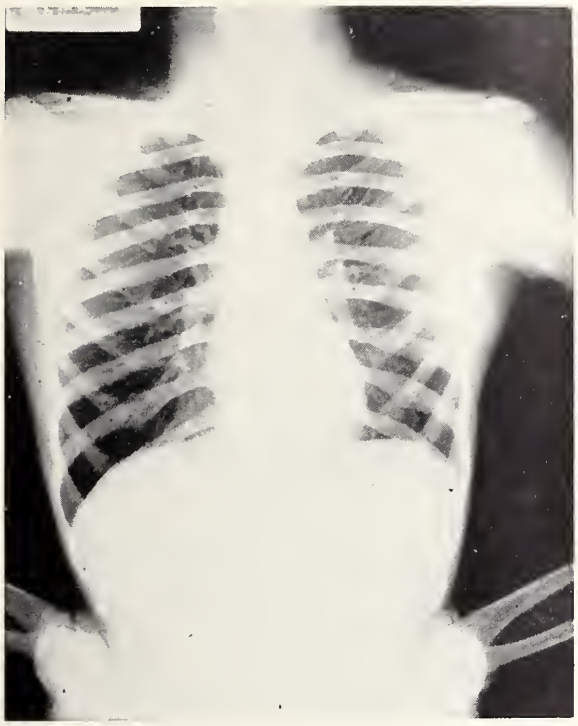


Figure 3

fluid. The mediastinum was in the midline, and the lungs remained well expanded.

The patient continued to make excellent progress. The thoracotomy tubes were removed on the second day, and the patient was ambulatory on the fourth day with normal pulse, temperature, and respiration.

The patient was discharged on the eighth postoperative day and returned to unlimited activity in three months.

A thorough examination of the patient was done nine months after his injury. An x-ray of the chest showed no abnormalities of the lungs or heart (Figure 3). Pulmonary function studies and electrocardiogram were completely normal. Angiocardiography demonstrated essentially a normal pulmonary arterial and venous pattern (Figure 4).

SUMMARY AND CONCLUSIONS

This case illustrates the fact that the exact nature of intrathoracic injury is unknown without thoracotomy. Therefore, the treatment of choice for traumatic hemothorax producing shock or cardio-respiratory embarrassment is immediate surgical exploration.

Interruption of a major pulmonary vein does not necessitate resection of pulmonary tissue inasmuch as the lungs have a large potential collateral circulation as demonstrated by Schlaepfer[1] and Swan.[2]

The preceding case demonstrates a complete return of normal cardio-respiratory function following interruption of a major pulmonary vessel.

BIBLIOGRAPHY

1. Schlaepfer, Karl: Collateral circulation in chronic obstruction of pulmonary vein and its relation to air embolism following various diagnostic and therapeutic procedures (pneumolysis), Surgery, Gynecology, and Obstetrics 37:510, 1923.

2. Swan, H., and Mulligan, R. M.: An experimental study of the effect of ligation of pulmonary veins in the dog, Journal of Thoracic Surgery 17:44-56, February, 1948.



Figure 4-A



Figure 4-B

Paroxysmal Ventricular Tachycardia Occurring During a Two-Step Exercise Test:

Report of Case

Robert A. Jordan, M.D.
Kansas City, Kansas

The following case is of interest because of the observation of a brief paroxysm of ventricular tachycardia which occurred in a patient during the performance of a double two-step test. This patient had complained of substernal discomfort rather infrequently after exertion. His symptoms were poorly described and were elicited only by leading questions; thus, it was the examiner's opinion that an electrocardiogram should be taken after having the patient perform an exercise test for the purpose of lending objective support to the diagnosis of coronary insufficiency.

From the Department of Medicine, University of Kansas Medical Center, Kansas City, Kansas.

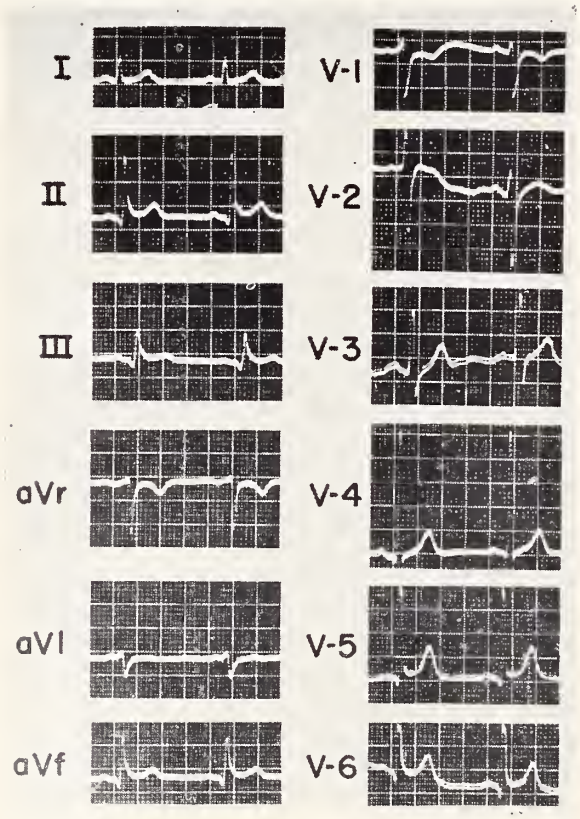


Figure 1. Electrocardiogram taken with the patient at rest.

Case Report

The patient, a white, married, professional man, 44 years of age, was first seen in the office July 28, 1952. He stated that his health was good but that he was accustomed to having an annual physical examination and had reported for this purpose. Further questioning of the patient revealed the fact that he had occasionally experienced a heavy, pressing sensation in the substernal region of his chest which was often associated with a vague, heavy discomfort in the left arm when he had climbed the 75 steps leading to his office. This discomfort was not severe and would subside quickly upon cessation of exercise.

In November 1949, the patient had experienced a sudden attack of syncope while participating in a square dance. A complete examination by a competent internist was performed on the following day, and no abnormalities were found. An electrocardiogram taken at the time appeared no different than the tracing shown in Figure 1.

Results of the physical examination and routine laboratory studies were normal. A roentgenogram of the chest was normal. An electrocardiogram taken with the patient resting is shown in Figure 1. The small Q waves observed in leads II, III, and aVF were not thought to be the result of any definite myocardial abnormality.

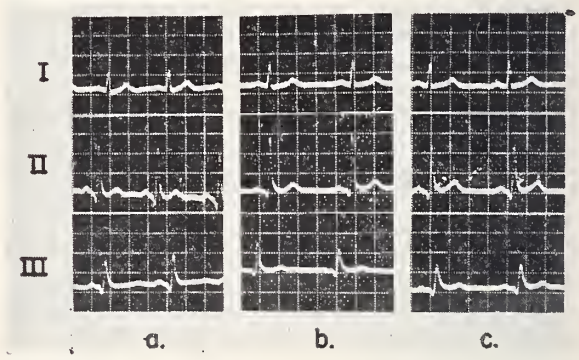


Figure 2. Electrocardiograms made (a) immediately after exercise, (b) 2 minutes after exercise, and (c) 5 minutes after exercise (20 complete trips over the two-step apparatus in 1 minute).

A second electrocardiogram (Figure 2) was taken after the patient had made 20 complete trips over the two-step apparatus in one minute. The results of this test were regarded as being within normal limits. The patient was allowed to rest for one hour, following which a third electrocardiogram (Figures 3 and 4) was taken after the patient had made 40 complete trips over the two-step apparatus in two minutes. The patient did not feel any substernal discomfort and was not aware of any irregularity of his heart beat either during or after this amount of exercise.

The electrocardiogram made immediately following exercise (Figure 3) shows several brief paroxysms of ventricular tachycardia in leads I and II. Some of the contractions in these same leads appear to be of sinus origin and show marked depression of the RS-T segments with inversion of the T waves.

The patient did not return for re-examination until October 27, 1953. During the interval between visits he had observed that his attacks of mild chest pain were neither more frequent nor severe than they had been. He stated that he had never been conscious of any irregularity of his heart beat. The resting electrocardiogram was repeated and appeared unchanged from the tracing shown in Figure 1.

COMMENT

The attack of syncope experienced by the patient while exerting himself strenuously at a square dance may have been the result of a sudden change in the cardiac rhythm, perhaps from a sinus rhythm to ventricular tachycardia. The patient did not give any history of other similar attacks of syncope, and he did not remember having noted any sensation of palpitation in his chest at the time of his attack.

Ventricular tachycardia is often a serious arrhythmia and is usually associated with severe organic

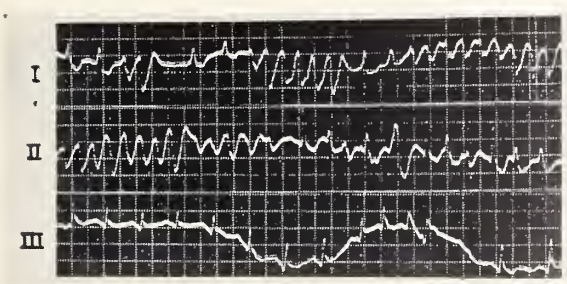


Figure 3. Electrocardiogram taken immediately after exercise (40 steps over the two-step apparatus in 2 minutes).

heart disease. This arrhythmia may often proceed into ventricular fibrillation and result in death. The occurrence of ventricular tachycardia during the performance of exercise tests has been reported by Klakeg and Parkin,[1] who have stated that such tests are not entirely without danger and that patients undergoing these tests should be carefully chosen and supervised during the procedure.

Master and associates[2] have stated that one of the criteria for an abnormal two-step test is the occur-

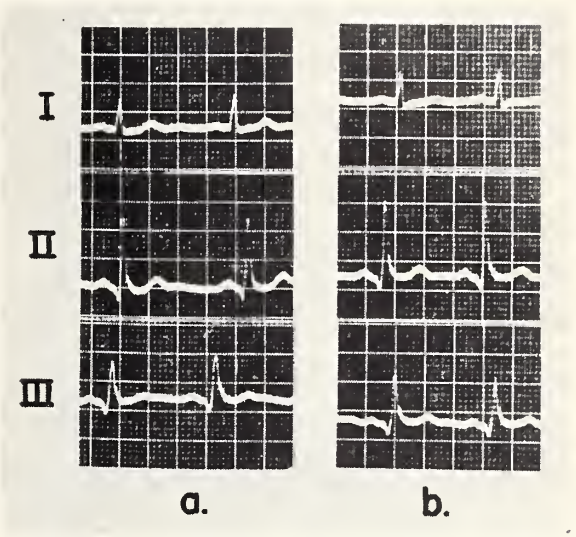


Figure 4. Electrocardiograms made (a) 2 minutes after exercise and (b) 5 minutes after exercise (40 steps over the two-step apparatus in 2 minutes).

rence of an arrhythmia following exercise. No evidence was presented to support this statement. Bourne[3] has observed, "Exercise would seem to diminish or leave unchanged the number of premature beats in normal and in rheumatic cases. Exercise would seem to increase their number in cases with evidence of arteriosclerosis or of coronary disease." There is evidence both for and against such a generalization.

Wenkebach and Winterberg[4] reported a case of a woman who gave a previous history of rheumatic fever and who suffered recurrent paroxysms of tachycardia following exertion. No evidence of coronary artery disease was present in this patient. There are several case reports[5, 6, 7, 8, 9] of patients having paroxysmal ventricular tachycardia induced by exertion, but in whom a careful examination of each patient gave no indication of the existence of organic cardiac disease.

Scott[8] reported on one patient who had complained of frequent attacks of ventricular tachycardia precipitated by exertion for more than 10 years but was not found to have any cardiac disease. Conversely, there are reports[1, 9, 10, 11, 12, 13] of several patients who experienced either premature ventricular contractions or ventricular tachycardia associated with angina pectoris following physical exertion. In some of these patients a daily ration of

quinidine appeared to increase their tolerance to exercise, not only by decreasing the occurrence of the arrhythmias, but also in some instances by reducing the frequency and severity of anginal attacks.[9, 11]

Klakeg and Parkin[1] have reported a case in which the two-step test induced angina pectoris and short paroxysms of ventricular tachycardia. They stated that this phenomenon had been observed previously in their laboratory.

Mann and Burchell[14] performed special studies on 21 patients in whom premature ventricular contractions had been observed electrocardiographically during two-step exercise tests. Their studies indicated that when premature ventricular beats are present in the resting electrocardiogram, an increase in the number of premature contractions induced by exercise did not seem to be correlated with the presence or absence of coronary insufficiency. In fact, their studies would seem to indicate that in those patients with no premature beats in the resting electrocardiograms, the induction of premature beats by exercise was closely correlated with definite clinical and electrocardiographic evidence of coronary insufficiency in each case. One interesting patient in their study with definite coronary insufficiency was found to have premature ventricular contractions in the resting electrocardiogram which were abolished by exercise.

It would appear from the reports reviewed that, in general, the induction of either premature ventricular contractions or ventricular tachycardia by exercise should be regarded as strongly suggestive of coronary insufficiency. At the same time, it is evident that there are fairly frequent exceptions to such a rule, which makes it desirable that additional evidence should be found in such patients before making conclusive diagnosis of coronary insufficiency.

SUMMARY

A case of paroxysmal ventricular tachycardia occurring during the performance of a double two-step test has been reported. The patient gave a history of angina pectoris occurring at times after exertion. In addition to the appearance of ventricular tachycardia in the electrocardiogram after exercise, there was also RS-T segment depression and T wave inversion in the few contractions which appeared to be of sinus origin.

BIBLIOGRAPHY

1. Klakeg, C. H., and Parkin, T. W.: Elevation of the RS-T segment in the precordial electrocardiogram and ventricular extrasystoles after exercise, Proc. Staff Meet. Mayo Clin. 29:97, 1954.
2. Master, A. M.; Friedman, R., and Dack, S.: The electrocardiogram after standard exercise as a functional test of the heart, Am. Heart J. 24:777, 1942.
3. Bourne, G.: An attempt at the clinical classification of premature ventricular beats, Quart. J. Med. 20:219, 1927.
4. Wenkebach, K. F., and Winterberg, H.: Quoted by Wilson,[9] et al., p. 163.
5. Anderson, M. C.: Paroxysmal ventricular tachycardia, Am. J. Med. Sci. 181:369, 1931.
6. Gilchrist, A. R.: Paroxysmal ventricular tachycardia, Am. Heart J. 1:546, 1926.
7. Hume, W. E.: Observations in six cases of paroxysmal tachycardia, Quart. J. Med. 11:131, 1918.
8. Scott, R. W.: Observations on a case of ventricular tachycardia with retrograde conduction, Heart 9:297, 1922.
9. Wilson, F. N.; Wishart, S. W.; MacLeod, A. G., and Barker, P. S.: A clinical type of paroxysmal tachycardia of ventricular origin in which paroxysms are induced by exertion, Am. Heart J. 8:155, 1932.
10. Goldhammer, S., and Scherf, D.: Quoted by Proger.[12] et al., p. 519.
11. Porter, W. B.: The probably grave significance of premature beats occurring in angina pectoris produced by effort, Am. J. Med. Sci. 216:509, 1948.
12. Proger, S. H.; Minnick, W. R., and Magendantz, H.: The circulatory response to exercise in patients with angina pectoris, Am. Heart J. 10:511, 1935.
13. Robinson, G. C., and Herrmann, G. R.: Paroxysmal tachycardia of ventricular origin and its relation to coronary occlusion, Heart 8:59, 1921.
14. Mann, R. H., and Burchell, H. B.: Premature ventricular contractions and exercise, Proc. Staff Meet. Mayo Clin. 27:383, 1952.

Historical Material Needed

In preparation for the observance of The Kansas Medical Society's centennial anniversary, members of the Committee on History are attempting to collect all material of historical interest. Physicians who can contribute information, records, etc., are urged to send such to

Committee on History
Kansas Medical Society
315 West 4th Street
Topeka, Kansas

Current Trends in Treatment of Congenital Megacolon

William N. Harsha, M.D.

Oklahoma City, Oklahoma

The history of true congenital megacolon is one of near misses in diagnosis and treatment for a 50-year period, only recently having been correctly evaluated. Even yet, the observations of the last few years fail to change the opinions of a large segment of the profession. It is possible now to understand congenital megacolon from genetic, pathological, and therapeutic aspects, these being the ultimate scientific goals in the study of any disease entity.

Congenital megacolon as a disease was first described in 1888 by a Danish surgeon, Hirschsprung, though megacolon in its various forms was known for centuries before.[1] It is unfortunate that in his classic description he was preoccupied with the dilated and hypertrophied colon rather than the probable cause of this dilatation. He only casually mentioned that the rectum was not dilated and perhaps some narrowed. His discussion centered on the marked hypertrophy of the bowel, especially the muscular layers, the inflamed and edematous mucosa, the ulcerations, and the submucosal abscesses. Subsequent observers, until recent times, followed Hirschsprung's lead and centered their investigations upon the dilated bowel and not upon the significance of the constricted rectum. An exhaustive review of these studies has been recently reported by Theodor Ehrenpreis, and further comment is not indicated here.[2]

Genetic observations of congenital megacolon are few in the literature and largely refer to isolated family histories. Most of these reports are based upon inadequate diagnostic (radiographic and pathological) criteria. A review of five genetic studies of congenital megacolon allows the following comments.[3]

Each observer felt that the material he had studied was not sufficient to draw absolute conclusions. They felt it was clear that there was a definite incidence of hereditary congenital megacolon. The strongest argument in favor of a familial incidence of the disease lies in the presence of megacolon in concordant monozygous twins as well as discordant dizygous twins. They found that uncles were not affected and felt this argued against sex-linked or sex-limited dominant characteristics. Likewise, they find no cases to follow a recessive genetic pattern. One author concluded that children of a parent who had congenital megacolon had a relatively slight chance of procreating a female sibling with megacolon, but one chance in five of having a male child with the disease.[7] In unaffected parents who have two siblings with megacolon, the chances of subsequent children with megacolon are high. The incidence of congenital megacolon is not high, occurring in one out of every 20,000-30,000 live births. It has been estimated that the average practitioner will see one case in his practice during his life.[8]

The differential diagnosis of congenital megacolon from organic obstructive lesions of the colon has been the source of present day knowledge of the pathological physiology of congenital megacolon. If the colon is pictured as being no different from any other hollow viscus of the body through which a regular flow must pass, the sequence of events peculiar to megacolon is clarified.

If the colon becomes acutely and completely obstructed, massive dilatation without hypertrophy will result, terminating in death if the obstruction isn't relieved. When the colon is chronically and incompletely obstructed, dilatation and hypertrophy of the colon wall will occur. When impaction of bowel materials above the site of obstruction occurs, pressure necrosis with mucosal ulceration and formation of submucosal abscesses result. If the obstruction is complete enough, this patient, too, will die the death of unrelieved bowel obstruction. In every case of megacolon it is essential to recognize that obstruction is present and to correct its cause. On this basis, megacolon can be divided into three groups.

1. *Aganglionic Megacolon:* Aganglionic megacolon, the true congenital form, is also called achalasic megacolon or Hirschsprung's disease. It is remarkably different from the other two variants. It was this variant that produced the confusion as to etiology up to recent years. Until the concept and subsequent proof of the absence of parasympathetic

This paper won the prize offered by the Kansas Chapter of the American College of Surgeons in its 1953 essay contest for interns, residents, and Junior Candidates of the College.

The author wishes to express sincere appreciation to Thomas G. Orr, Sr., M.D., and Stanley Friesen, M.D., for invaluable suggestions and technical assistance in the preparation of this paper.

ganglion cells in the terminal colon segment, no apparent cause for the disturbance was recognizable. Cameron was the first to present this ganglionic absence hypothesis.[9]

Hiatt later described the physiological defect that attended this ganglionic absence.[10] In his work, by use of balloon and kymographic studies, he has shown that a type of mass contraction occurs in such an achalasic colon but that no orderly peristalsis occurs. The result is a functional, though not a mechanical, obstruction allowing little more than overflow to pass. The natural fecal stream of the lower colon, being firm and semisolid in consistency, demands a highly efficient peristaltic action for passage to the anus.

Hiatt's work substantiates the pathological observation that the area of ganglionic absence rarely extends more distally than the lower rectum nor proximally past mid-descending colon.[4, 12, 13] Regardless of the level of ganglionic absence, this, too, is the level of physiological obstruction. The fact that the ganglia again appear in the lower rectum and anus explains the characteristic picture of obstruction at the anus.

The diagnosis of this megacolon variant is not difficult. Megacolon history from birth with physical signs of dilated colon on abdominal examination is typical. The rectal examination shows a narrowed anus with a rectal ampula, expanding into a widely dilated viscus containing inspissated material. Barium enema, especially using thin barium and utilizing oblique views, will demonstrate a widely dilated colon extending proximally for a varying length and tapering to a point of apparent obstruction in mid or lower rectum. Grossly, the cleansed colon appears normal to examining finger and proctoscopic examination. The universally consistent observation in congenital megacolon is that the obstructive symptoms have their onset at birth or within the next few days. These symptoms can be relieved temporarily by conservative means but never completely or permanently. The early case of aganglionic megacolon that has received conscientious conservative therapy will be difficult to diagnose owing to the lack of sufficient mechanical obstruction to produce the typical physical and roentgenological signs. In this situation the history is generally pathognomonic.

A significant number of patients with absence of myenteric plexi likewise present a deficiency or rarely an absence of parasympathetic ganglia in the renal pelvis, ureters, urinary bladder, or urinary sphincters. Hydroureter or massive urinary retention with attendant infectious complications is frequently present in these patients. Surgical correction of megacolon obviously does not affect the course of these urinary situations.

2. *Functional Megacolon:* Functional or idiopathic megacolon represents the situation where fecal impaction is present at the anus and allowed to persist. Most authors feel it has its origin in improper bowel habit and diet or is associated with emotional problems. Anal fissures are known to produce or to aggravate this situation. A small percentage of these cases represent the congenital elongation of the colon described by Martinotti as well as Jones and Morton.[14, 15] This elongated colon, or dolichocolon, is believed to absorb an excess of fluid from the bowel residue producing a mass which is so inspissated that normal colonic peristalsis cannot advance it to the anus. Satisfactory results have been reported from surgical extirpation of this type of dolichocolon.[15]

These cases are diagnostically differentiated in that they do not commence at birth. Also there is no digital or x-ray evidence of obstruction at the rectal ampulla.

3. *Organic Megacolon:* Most cases of megacolon belong to this variant which always has an organic obstructive lesion as its basis. The common lesion is the imperforate anus or postoperative cicatrix at the site of imperforate anus. Intrinsic and extrinsic tumors as well as less frequent congenital anomalies constitute most of the remaining obstructing lesions.

The treatment of megacolon has passed through many fads and only in recent years has it been successfully managed. Most important is the establishment of the real cause of megacolon, be it due to deficiency of myenteric plexi, megacolon secondary to functional disease, or due to an organic obstructing lesion.

Numerous surgical and medical plans have been attempted in the aganglionic type of megacolon. Repeated enemas will afford temporary relief. An occasional fatality due to water intoxication associated with profound potassium depletion has been produced by such repeated enemas.[10] This can be avoided by the use of isotonic solutions, such as normal saline or gelatin.

An early lead to the real cause of aganglionic megacolon was suggested, though not realized, shortly after Hirschsprung's classic description. A British surgeon, Sir Frederick Treves, performed a colostomy for what appeared to be aganglionic megacolon. The colostomy did not function, and he performed a proctosigmoidectomy with construction of a perineal colostomy. This gave a physiologically successful though impractical result.[16]

After this experience, colostomy continued to be the accepted treatment until recent years. Usually colostomy will offer symptomatic relief, but the megacolon invariably recurs when the colostomy is closed. Thus the need for a better procedure is ap-

parent. Some 15 years ago interest in this field was focused upon the use of parasympathomimetic and sympatholytic drugs. Not a small number of internists felt this was the answer, and many support this management yet.[14, 17, 18, 19]

The current trend in treatment is surgical extirpation of that section of the colon which is devoid of myenteric plexi and reconstruction of the colon so that a functioning anal sphincter is preserved. Swenson and Bill were the first to describe this technique, and universal success has followed.[20] An important refinement in the procedure was shown by Hiatt and was the procedure followed in the author's cases.[21]

The surgical procedures described by Swenson and Bill and modified by Hiatt constitute a proctosigmoidectomy with end to end anastomosis leaving a functioning anal sphincter. The patient first receives bowel preparation designed to remove as much fecal residue as possible and to render remaining colon contents bacterially sterile.

The surgical technique is undertaken much as is a combined abdominal-perineal resection of the rectum. First the patient is placed supine on the operating table, and the abdomen is entered through a generous "hockey stick" incision extending from pubis to navel in the left paramedian line and thence laterally to a point just below the tip of the left 12th rib. The proximal point of colonic resection is chosen, generally, in the mid-descending colon, and the mesenteric blood supply from this point distal to the pelvic peritoneal reflection is incised with vessels being ligated.

The proximal descending colon, splenic flexure, and transverse colon are mobilized so that the point of resection will fall easily into the pelvis without compromise of its blood supply. Pelvic peritoneum is then incised, and the recto-sigmoid and rectum are freed from the sacral concavity and sharply dissected from the anterior pelvic structures. In the male, care must be taken to avoid prostate and prostatic urethra. The rectum is freed to the level of the levator ani muscles.

Prior to the pelvic dissection, identification of the ureters on both sides must be accomplished. The patient is then placed in lithotomy position, and an assistant passes an instrument through the dilated anus into the lumen of the rectum. The instrument is then guided by the surgeon through the rectum to the point of resection in the descending colon. The clamp is used to grasp the interior of the colon at this point and is slowly withdrawn by the assistant, thereby intussuscepting the colon at the point of resection into the freed recto-sigmoid and rectum through the anus and externally through the anal sphincter. The exteriorized colon is partially resected, exposing the serosa of the colon at the point of its resection.

A layer of sutures approximating the serosa of the colon to the pelvic muscularis of the anus is placed, followed by a second layer approximating the colonic and anal mucosa. The balance of exteriorized bowel is resected, the suture layer of colonic serosa to the anal muscularis is completed. Finally the layer approximating the colonic to the anal mucosa is finished. This makes a two-layer end to end anastomosis between the anus and the mobilized colon. The suture line is reduced past the anal sphincter, appropriate perineal drainage is established, and the patient is again placed in supine position. Most surgeons prefer to leave a soft rubber rectal tube inserted past the suture line. Redraping is accomplished. The pelvic peritoneum is reapproximated and the abdomen closed in usual fashion. Postoperative management is no different from any colonic resection.

A review of recent literature shows that more than 100 cases of aganglionic megacolon have been treated by these recent surgical techniques, and each author reports uniform and apparently lasting success.[21, 26]

The treatment of the patient diagnosed as having functional megacolon is frequently a frustrating problem. It is best dealt with by the internist and the psychiatrist and only rarely presents any surgical problem.[14] The unusual case of dolichocolon has been previously referred to.[15] Management of the third group is usually obvious in the removal or correction of the obstructing organic defect. An excellent review of the problems met with in this group is found in a recent symposium edited by Ravitich.[25]

In all cases where aganglionic megacolon is suspected, a complete urological survey should be performed to ascertain whether the ganglion deficiency is likewise present in the ureters, urinary bladder, or sphincters.

CASE REPORTS

Case 1. S. P. was a white female, full term normal delivery, oldest of five siblings. Her first hospital admission was on November 6, 1941, when she was seven weeks old. She was admitted with the chief complaints of diarrhea, abdominal distention, and marked dehydration of four days duration. She gave a history of no normal bowel movements since birth except for passage of what was described as meconium during the first three days of her life. Treatment during this admission was parenteral fluids, phenobarbital, and repeated enemas. Diagnosis was fecal impaction.

Her second admission was May 8, 1942, at the age of seven months. The chief complaint was vom-



Figure 1. Thin barium, oblique x-ray studies of colon showing point of tapering constriction at lower recto-sigmoid and marked dilatation recto-sigmoid and descending colon.

iting and obstruction of the lower bowel. She gave a history of many episodes of vomiting since birth, each attendant to marked abdominal distention, and each following abandonment of the prescribed daily enemas. She had had no normal bowel movements since birth. Barium enema at this time showed fecal impaction. She was treated with digital removal of the impaction and enemas and was discharged on daily oil retention enemas and petrolagar.

The patient was not seen again until July of 1951 when she was nine years old. Her history at this time was one of life-long constipation with frequent fecal impactions. It was possible to maintain her only partially free from marked abdominal distention with episodes of vomiting, accomplished by use of repeated daily enemas. The child was markedly emaciated, lethargic, and chronically debilitated.

On physical examination, the abdomen was found to be distended, and it was possible to palpate the transverse and descending colon as a widely dilated viscus containing doughy material. It was not possible to elicit peristaltic motion. Rectal examination showed no abnormality of the sphincter or anus, and the tip of the examining finger entered a widely dilated, stool-containing viscus. General physical examination revealed no other pathology.

X-ray showed marked megacolon with suggestive stricture at the lower end of the rectum (Figure 1). Sigmoidoscopy showed a tight anal sphincter and anus without stricture which passed abruptly into a widely dilated recto-sigmoid containing a large amount of inspissated stool. The mucosa appeared normal throughout. Intravenous pyelograms were normal. Laboratory examinations revealed moderate microcytic anemia, and the serum protein was in the lower limits of normal. Urine examination showed no evidence of kidney disease or urinary infection.

The patient was felt to have congenital aganglionic megacolon, and a Swenson type proctosigmoidectomy was recommended. She was placed on a bowel preparation of cathartics, daily enemas, and wide range antibiotics. On July 24, 1952, a Hiatt modification of the Swenson-Bill prostosigmoidectomy was performed.

At surgery the descending colon, recto-sigmoid and upper rectum were markedly dilated and hypertrophied (Figure 2). The transverse and ascending colon were dilated moderately. In the mid-rectal area, 3 centimeters below the peritoneal reflexion, an area of apparent constriction was demonstrated. Pathologically, the area from the mid-descending colon to the area of constriction in the mid rectum



Figure 2. Surgical specimen showing dilated and hypertrophied descending and recto-sigmoid colon with constricted area (upper right hand corner) at lower end of recto-sigmoid.

showed complete absence of myenteric plexi (Figure 3). Her postoperative course was uneventful. She was discharged on the 10th postoperative day, having frequent soft stools and nearly complete remission of abdominal distention.

She was readmitted once in February and again in March of 1953 with complaints of urinary frequency. Complete urological work-up on each admission showed no evidence of urinary pathology. The possibility of deficiency of myenteric plexi in the ureter or urinary bladder was considered but never verified. During the last two admissions she reported having daily formed stools and no episodes of abdominal distention or vomiting. Her physical examination was one of marked increase of tissue turgor, 10 to 12-pound weight gain, and a remarkably increased sense of well being.

In November of 1953 examination of the patient revealed an apparently normal adolescent girl. No abdominal distention was present, and she reported daily normal bowel movements. There have been no further urinary complications.

Case 2. M. W. is a five-year-old colored male, eldest of three children. History reveals a spontaneous normal delivery of an eight-pound two-ounce infant. At two weeks of age the baby had had only one meconium stool, and he was given an enema

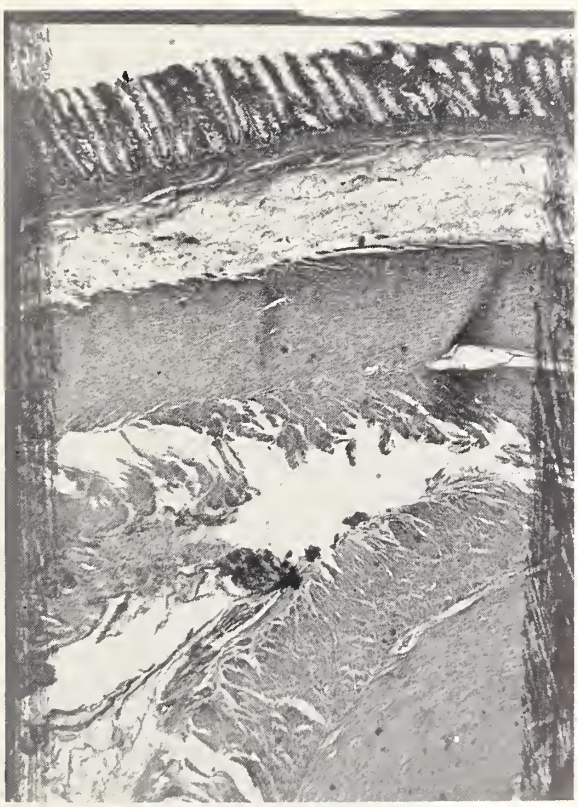


Figure 3. Microscopic section taken 4 centimeters proximal to recto-sigmoid constriction. Note complete absence of myenteric plexi.

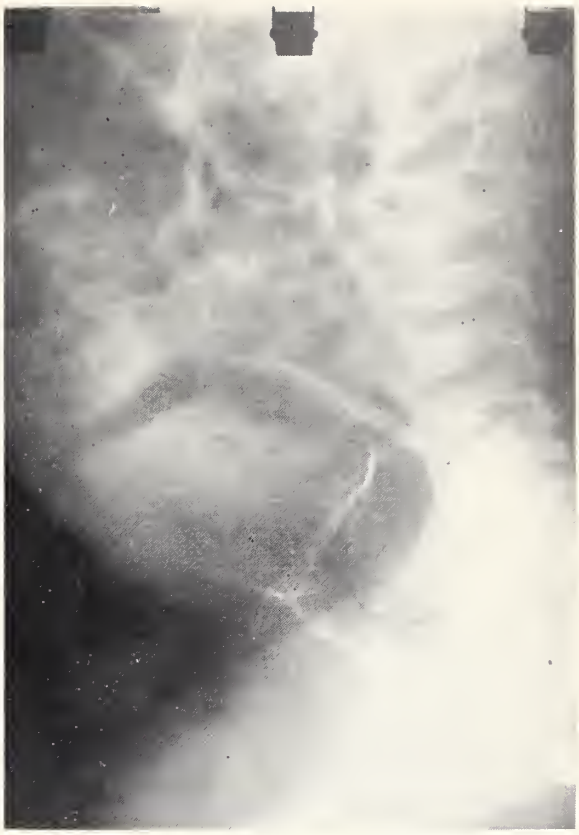


Figure 4. Thin barium, air contrast, and oblique x-ray showing point of constriction at upper rectum.

with large results. During his first year his only bowel relief was afforded by repeated enemas. From the age of one to four years the patient occasionally developed diarrhea which persisted for three to ten days. It was only during these periods that the marked abdominal distention, always present since birth, receded. The mother stated that invariably the patient felt and played better during the episodes of diarrhea than the "normal" times. It was necessary to administer daily multiple enemas to prevent him from distending to a point where he refused to eat or became nauseated. During his fourth year of life the patient experienced three episodes of profound abdominal distention unrelieved by enemas, vomiting, and extreme lethargy. The patient was hospitalized each time, received "fluid" under his skin and "shots," and responded later to high enemas.

At the time of my first examination, a chronically ill, emaciated colored boy, five years old, was presented. He weighed 27 pounds and was markedly lethargic. General physical examination revealed only a markedly distended abdomen in which the ascending, transverse, and descending colon could easily be outlined. Occasionally discordant peristaltic waves could be seen or elicited by stroking.

The dilated bowel contained a tremendous amount of doughy material.

Rectal examination revealed a normal anus with a constricted area some 3 centimeters from the anus that would admit the index finger with difficulty. Proximal to this, the examining finger entered a smooth dilated viscus, packed with inspissated material and lined with what felt like normal colonic mucosa.

Laboratory findings revealed a hemoglobin of 9.5 grams and a normal white blood and differential count. Blood proteins were 4.2 grams with albumin being 2.8 grams. Other blood chemistry was within normal limits.

The patient was hospitalized, placed on multiple cleansing enemas of normal saline, and subsequently sigmoidoscoped. No organic lesion was demonstrated. The anal canal was 3 centimeters in length and emptied abruptly into dilated bowel, which still contained large amounts of stool. Barium enema with thin barium and multiple oblique films revealed a typical tapered cut-off at the upper anus with markedly dilated colon proximally to and including the caecum (Figure 4).

It was felt that the patient presented the typical picture of aganglionic congenital megacolon, and a Swenson type operative extirpation of the involved colon was recommended. The patient was placed upon intensive high protein feeding, whole blood transfusions, wide scope antibiotic therapy, repeated daily saline enemas, and cathartics. On the date of operation the hemoglobin was 12.8 grams, and the blood protein was 7.2 grams with 3.9 grams of albumin. Upon entry into the abdomen, the colon was found to be markedly dilated and hypertrophied to the mid transverse colon with moderate dilatation extending into the caecum. An area of marked hypertrophy of the colon wall extended from the mid-descending colon to an area of constriction at the upper level of the anus (Figure 5). The Hiatt modification of the Swenson operation was performed. Pathologically there was an absence of myenteric plexi from mid colon to upper anus (Figure 6). The postoperative course was uneventful.

Semi-liquid stools were passed from the fifth to the tenth days, followed by progressively formed stools. Postoperative check visits show that four months after surgery the patient had one or two formed stools daily, ate lustily, weighed 40 pounds and was no different from his playmates in activity. His hemoglobin remained over 13 grams and blood protein over 7.3 grams. He had had four episodes of diarrhea, each occurring during influenzal outbreaks in his home, and each readily controlled.

BIBLIOGRAPHY

1. Hirschsprung, H.: Jahrb Kinderheilk 27:1:(1888).
2. Ehrenpreis, T.: Acta Chir. Scandiv. 94:112:46.
3. Dalle Valle, A.: Peditricia 28:74:24 and 32:569:24.
4. Zuelzer, W. W., and Wilson, J. L.: Amer. J. Dis. Child. 75:40:48.
5. Buttersack, P.: Münch Med. Wschr. 74:1626:27.
6. Popper, J.: New York Med. J. 112:1030:20.
7. Bodian, M.; Carter, C. O., and Ward, B. C. H.: Lancet 160:302:51.
8. Conole, F. D.: New York State J. Med. 51:2419:51.
9. Cameron, J. A. M.: Arch. Dis. Child. 3:210:28.
10. Hiatt, R. B.: Ann. Surg. 133:313:1951.
11. Hightower, N. C.; Code, C. F., and Maher, F. T.: Proc. Staff Mayo Clinic 24:453:49.
12. Tiffen, M. E.; Chadler, L. R., and Faber, H. R.: Am. J. Dis. Child. 50:71:48.
13. Whitehouse, F. R., and Kernohon, J. W.: Arch. Int. Med. 82:75:48.
14. Lee, C. M.; Begg, K. C., and Brown, J. R.: Surg., Gyn. & Obs. 91:281:50.
15. Jones, T. B., and Morton, J. J.: Amer. J. Surg. 39: 382:38.
16. Treves, F.: Lancet London 1:276:(1898).
17. Grimson, K. S.; Vandergrift, H. N., and Dratz, H. M.: Am. J. Dis. Child. 68:102:44.
18. Crile, G. (Jr.), and Nosik, W. A.: Surg. Clinic N. Amer. 19:1163:39.
19. Rankin, F. W., and Learmenth, J. R.: Ann. Surg. 92: 710:30.
20. Swenson, O., and Bill, A. H., Jr.: Surgery 24:212:48.
21. Hiatt, R. B.: Am. Surg. 133:34:51.
22. Swenson, O.; Neuhauser, E. B. D., and Pickett, L. K.: Pediatrics 4:201:49.
23. Rosin, J. D.; Barger, J. A., and Waugh, J. M.: Proc. Staff Mayo Clinic 25:710:50.
24. Swenson, O.: Surgery 28:371:50.
25. Ravitch, M. M.: S. Clin. N. Amer. 29:1535:49.
26. Evans, C. H.: Surg., Gyn. & Obs. 92:1:51.

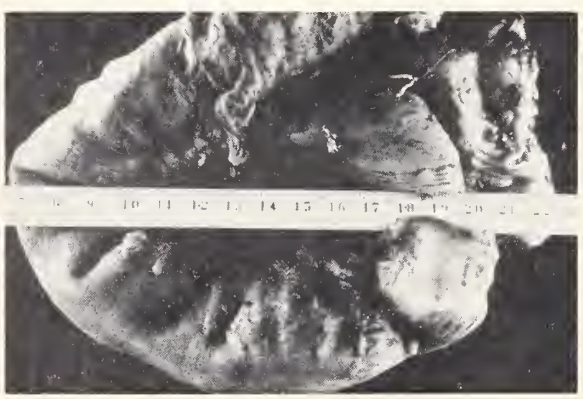


Figure 5. Surgical specimen showing dilated and hypertrophied descending and recto-sigmoid colon narrowing to a point of constriction in upper rectum.



Figure 6. Microscopic section taken at recto-sigmoid junction and showing complete absence of myenteric plexi.

THE MONTH IN WASHINGTON

Editor's Note. The following summary of Washington news was prepared by the Washington office of the A.M.A. for distribution to state and regional medical journals.

When the 84th Congress convenes in January, the Eisenhower administration will press for passage of at least two bills that failed to get through last session, reinsurance and a new program of medical care for military dependents. The former was decisively defeated in the House. The latter did not reach a vote in either chamber.

In a radio address summing up his administration's legislative achievements, Mr. Eisenhower confirmed that he was prepared to renew the fight next session to have the federal government set up a system for reinsuring health insurance programs. He declared: "Health reinsurance we are going to put before Congress again because we must have a means open to every American family so that they can insure themselves cheaply against the possibility of catastrophe in the medical line."

There have been no indications how far the administration would go in amending the reinsurance bill to satisfy its critics. It is possible also that if all objectionable features were removed there would be little left of the bill.

At Senate and House hearings, reinsurance was roundly denounced by most witnesses, for a variety of reasons. A.M.A.'s position was that reinsurance wasn't needed because private funds are available for the limited amount of reinsurance that could be used, and that in addition the program projected the federal government too far in the direction of control of medical care.

Later in the session, Mr. Eisenhower himself and Mrs. Hobby made every effort to win over critics of reinsurance, and to force the bill through Congress. In the light of these efforts—including a nationwide radio appeal by Mrs. Hobby—the defeat of the bill in the House of Representatives was regarded as one of the most surprising suffered by the administration on any domestic legislation.

Currently Secretary Hobby and Chairman Charles Wolverton of the House Interstate and Foreign Commerce Committee are attempting to bring together all parties interested in health legislation to see if a compromise can be worked out on reinsurance.

Although the dependent medical care bill wasn't passed, this fact was not in any way regarded as a defeat for Mr. Eisenhower. The bill was offered in the Senate in plenty of time for action, but the introduction of the House bill was held up until Defense Department could estimate the first year's cost, eventually set at $67 million. At any rate, neither Senate nor House Armed Services Committee held hearings on the measure.

In another statement, Mr. Eisenhower made it clear that he expects the next Congress to do something about improving and making more uniform the system of medical care for servicemen's families. Congress, he said, "must eventually meet certain imperative needs of the members of the armed forces." He explained that servicemen now "lack adequate medical care for dependents. . . . It is most important that these needs of the armed forces personnel serving their country, often in remote corners of the world, engage our serious consideration."

Although the A.M.A. has not had an opportunity to testify on the dependent care plan before Congressional committees, it has made its views known to the Defense Department. In general the A.M.A. is not opposed to Defense Department proposals that a more uniform system be worked out, and that the federal government bear most of the cost. On one important point, however, the recommendations of the department and of the A.M.A. are in direct conflict. The department would have the military medical departments themselves furnish dependent medical care wherever they could, with service families going to private physicians and private hospitals only where the uniformed physicians couldn't handle them. The A.M.A., on the other hand, proposes that dependents be cared for by military medical departments only where civilian medical facilities are inadequate to furnish proper care.

Federal officials, meanwhile, are busy preparing to put into effect the new health bills passed by Congress. Basic state allotment percentages have been worked out for the new Hill-Burton program (for facilities other than complete hospitals) and for the expanded vocational rehabilitation program. The Internal Revenue Bureau is about to issue detailed instructions to taxpayers regarding changes in medical expense deductions and other benefits in the new tax law.

Several large contributions from state medical associations have boosted the American Medical Education Foundation nearer its goal of two million dollars in 1954.

The California Medical Association recently contributed $100,000. Another large contribution came from Arizona in the amount of $7,230, representing $10 per member.

PRESIDENT'S PAGE

Dear Doctor:

Previous presidents have commented upon the committee effort conducted by this Society, and even with some knowledge of the subject I have been impressed with the scope of such activity. Some committee is in session on almost any Sunday of the year, and a few weeks ago three were meeting simultaneously.

Attendance at these meetings is surprisingly good. Members come from all over the state, not only giving up their time but incurring some considerable expense in this effort. Discussions are serious and thoughtful and constructive. Nearly every progressive project undertaken by the Society is the product of committee planning, which this year again will be demonstrated in action.

Even that is not the most surprising fact. The wide contact of these committees with other organizations makes their influence extend far beyond the confines of our membership. Groups meet with representatives of other professions, with a variety of business leaders, with governmental agencies, and with voluntary health associations. Through this united effort, contacts are made that are of inestimable value to each of us.

The only disturbing factor in this otherwise completely bright picture is that so much of our productive effort is contributed by so few. As your president I urge each member to participate in these activities. You are welcome to attend any meeting at any time. You will be placed on any committee on which you wish to serve, or your ideas for committee activity will be most gratefully received. I would be pleased to hear from you.

Yours,

Murray C. Eddy, M.D.

EDITORIAL COMMENT

Maternal Mortality Study

July 1, 1954, marked the beginning of a new method for the study of maternal mortalities in Kansas. This program will be executed by the combined efforts of the Kansas State Obstetrical Society, the Maternal and Child Health Division of the Kansas State Board of Health, and the Committee on Maternal Welfare of The Kansas Medical Society.

The study of maternal mortality has long been one of the primary functions of the Maternal Welfare Committee. Although the study has promoted some interesting discussions in numerous meetings, the committee has felt that the study was deficient in several respects. The maternal mortality questionnaire in the past has been inadequate in scope and execution. The long delay between the maternal death and the study by the committee resulted in the loss of considerable information which would have influenced the assignment of responsibility. Interesting and educational points evolving from the study were not applied in a manner to improve obstetrical care or promote obstetrical education. The present study, it is hoped, will correct all of these deficiencies. Deaths occurring during pregnancy, within three months after delivery, or at a later time if the pregnancy might have been a factor in causing the death, will be included in the study.

A completely new and expanded questionnaire will be used in obtaining information about each maternal death. This questionnaire has been adopted following examination of several in use in other states and is patterned after that currently in use in Minnesota. The members of the committee have no greater love for filling out voluminous forms than their fellow practitioners, especially when the subject matter pertains to the unpleasant facts of a medical tragedy. It is thought, however, that information obtained from this type of questionnaire warrants the full co-operation of the committee as well as the attending physician. Therefore, it has been agreed that a member of the Kansas State Obstetrical Society will be designated to contact personally the attending physician in each case and assist him in the completion of the questionnaire. Members of the Maternal Welfare Committee have volunteered to assume this task in earlier phases of the study.

The loss of time in initiating the questionnaire in any particular case diminishes the total information and subsequent value of the study. The committee is requesting that maternal deaths be reported immediately to the chairman of the committee or to the Maternal and Child Health Division of the Kansas State Board of Health. This, of course, does not preclude the necessity of completing the official death certificate; it is merely a means of transmitting information about the death to the interested parties as quickly as possible.

The chairman will contact a member of the committee and request his co-operation in completing the questionnaire. The questionnaire then will be submitted to the committee without identification of the attending physician. The case will be discussed as such cases have in the past and, from the discussion, assignment of responsibility will be made and any points of educational value will be developed. The physician involved will be advised of the committee's opinion in the hope that it may offer him guidance for future problems.

Cases of unusual interest or instructive value will be discussed anonymously at meetings of the Kansas State Obstetrical Society. It is planned that—again anonymously—abstracts of these cases may be published in the JOURNAL for purpose of wider distribution of the information.

Members of The Kansas Medical Society who attended the annual meeting in Topeka in May had the opportunity to see a graphic presentation of the plan prepared through the co-operation of the interested groups. It is planned that each physician in the Society shall receive a copy of the questionnaire for his perusal in the near future.

The aim of the committee in pursuing this study is the development of better maternal care. It feels that no maternal death can occur without presenting some element of information of value in the management of the health of a pregnant woman. It is hoped that any physician contacted in such a case will accept the study in the spirit intended, not necessarily as a criticism or disciplinary procedure.

Federal Aid in Kansas

PART I

During the months to follow, the JOURNAL will publish a series of articles discussing federal aid for programs operating in this state. A brief series concluded in September recorded federal expenditures for health. This is related but projects the discussion in a different direction. Here will be listed not only health but all federal participating programs as they operate in this state.

The material is taken from two sizable reports. The first is a 50-page mimeographed "Tentative Report of Kansas Commission on Federal-State Relations." This was created by Gov. Edward F. Arn on October 13, 1953, in co-operation with the Pres-

ident's Commission on Intergovernmental Relations. The second is a 44-page report by the Kansas State Chamber of Commerce entitled "Basic Summary of Federal Grant-in-aid Programs in Kansas 1953."

The JOURNAL proposes this to be factual and will make no comment on any program here described except to quote from the above-mentioned reports. The purpose of this series is to acquaint The Kansas Medical Society with Kansas projects that have federal money and to describe how this money is received and disbursed.

It should be mentioned that figures will indicate money actually spent in Kansas, rather than how much was allocated. A further point of explanation concerns the difficulty of accurate classification because of overlapping and differences in policy. For example, the Superintendent of Public Instruction is responsible for the school lunch program, but the Board of Social Welfare reduces the cost by distributing surplus federal foods, which in turn affects farm price supports. Then, in another way, the Board of Health is also involved.

The story described in this series will be as accurate as possible without resorting to almost endless minor exceptions and qualifications. Should further information be desired, this may be obtained by writing the JOURNAL.

In 1953 Kansas participated in 35 federal grant-in-aid programs. Some $41,500,000 federal funds were received and spent by 13 separate state agencies. In the use of this money, Kansas spent $25,000,000 from the state treasury, and local governmental units spent at least $16,000,000. This last figure is frequently not available, so the total is actually larger than listed. It is correct to say that Kansas matched from state and local accounts everything the federal government gave even though the matching formula varies widely.

As will be shown in subsequent issues, the degree of federal control varies also. In some cases only a co-operative agreement is required. Other federal grants are made upon rigid stipulations which must be adhered to.

On January 30, 1954, the Kansas Commission adopted six basic principles as guides for future recommendations. These, while not strictly within the scope of this study, may show Kansas thinking with reference to the over-all subject of federal aid. At least, it is the thinking of the 15 men and women representing the governor's commission.

The first is that the cost of government will continue to rise at all levels unless there is a halt in establishing new government operated or subsidized programs and unless overlapping and duplication are adjusted. Second, the Commission sees its effort as most effective in the correction of overlapping services.

Third is the belief that correction of these conditions can best be achieved if a single level of government will collect the revenue for and administer the operation of a specific program. Co-operation between the various levels of government should not depend upon financial considerations so that federal aid will cease to be federal control.

Fourth is the recognition that such changes cannot be made hastily but that the gradual realization of these basic aims will prove the best course of action.

Fifth is that the Commission does not desire to curtail existing functions that are of service to the progress, health, welfare, or education of Kansas but that it hopes to reduce wasteful operations and expenditures.

Sixth, "We believe that the trend of centralization and financial domination by the federal government weakens the state and local government and that a reversal of this trend will not only strengthen the state and local governments, but will make a stronger national government."

The next article in this series will discuss federal grant-in-aid projects in Kansas relating to agriculture. Subsequent subjects will be education, health, highways, welfare and other programs.

UNITED FUNDS

There is a movement abroad in the land which physicians might well take note of. It has various names, United Fund being the most common. The philosophy is simple. Since there are so many fund campaigns of various sorts, the simple thing seems to be to lump all fund drives into one super fund for all causes.

Admittedly, the problem is complex, but the solution is not. Let's examine some of the facts, both pro and con. On the pro side of the ledger are:

1. Simplification of the fund-raising process, with easy supervision of the entire structure by a central budget and planning committee.

2. Fewer workers having to give time again and again to the various agencies.

3. Greater public acceptance (so it is claimed), since there is theoretically only one giving per year.

4. Lower campaign costs (either by having professional fund-raising guidance or by combining into one structure).

Here are some of the arguments against the single United Fund:

1. Loss of identity of the participating agencies. The people who raise the money are the ones most interested in its expenditure.

2. Rapid leveling off of the top limit of income, which is set by the budget committee of the Fund. Agencies with a relatively large financial potential are soon hamstrung by an inability to grow. This is particularly true of the health agencies which are just now beginning to burst forth with a real flower. You know what research means to the health of this country. It's expensive, but is, of course, the ultimate answer.

3. The United Fund takes away the very vital right of choice of the donor. Individual agencies are founded on the premise that the public will invest in research and good health, and the resultant loss of the right to choose can and does stifle growth.

4. Most national agencies have their own campaign structure and can conduct a campaign without exorbitant costs. When the total income of agencies campaigning outside United Funds is taken, the same percentage is used for campaign (since they all make much *more* in their own drive).

5. The trend toward centralization of fund-raising is one more step toward a centralized control which is undesirable. Where does it stop? The government?

6. By what right do a few persons in a community take it upon themselves to regulate giving? Whose right is it to tell a donor how much he must give? (The common *demand* of United Funds is one and one-half days pay. How many can or will part with that much at one swoop? How many will give much more to various agencies, if spread over a period of months?)

Further, what right does a small, select group in a community have to regulate the budget of any agency, when the board of directors of that agency gives a great deal of time and effort in concentrated study of the specific problem? Who is qualified to judge matters—especially in the health field—the laymen on the local United Fund board, or the board of directors of the agency? In all of the health agencies, Kansas physicians play a prominent role in guidance. Most have doctors of medicine for their president.

As mentioned, the problem is complex. It appears that members of the medical profession, by exercising their rights as citizens of a community, can help bring an enlightened answer to the problem. Time waits for no man, and the United Fund movement is gaining momentum. Now is the time, if your town or county is planning a United Fund, to take action.

Gerald L. Shannon, Executive Secretary
The Kansas Heart Association

SERVICE SEPARATIONS

As a service to physicians and communities in this state desiring additional medical personnel, the Journal of the Kansas Medical Society will publish in this column each month the names of medical officers who will shortly be separated from the armed forces. These are men who volunteered from Kansas, and many of them will probably be interested in finding locations in this state. Anyone interested in contacting these physicians may write to the address here given.

John S. Anderson, M.D.
814 Greenway Court
Wichita, Kansas

Lewis George Allen, M.D.
3137 North 54th Street
Kansas City, Kansas

Robert S. Brown, M.D.
5009 51st Terrace
Mission, Kansas

George G. Browning, M.D.
316 South 8th Street
Humboldt, Kansas

Dean C. Chaffee, M.D.
417 N. W. Third
Abilene, Kansas

Charles A. Crockett, M.D.
5437 Neosho
Kansas City, Kansas

Raymond G. Elliott, M.D.
6136 Reinhardt Drive
Mission, Kansas

Tex Elholm Fury, M.D.
425 Putnam Avenue
Salina, Kansas

Henry S. Haerle, M.D.
Marysville, Kansas

Wayne E. Hird, M.D.
102 North Byron Road
Wichita, Kansas

Robert A. Jelinek, M.D.
506 Chestnut Street
Leavenworth, Kansas

Earl G. Padfield, Jr., M.D.
4412 West 70th
Prairie Village, Kansas

Perry D. Petterson, M.D.
3717 East Murdock
Wichita, Kansas

Robert L. Satterlee, M.D.
Macksville, Kansas

William C. Swisher, M.D.
2920 Sennett
Wichita, Kansas

Robert E. Young, M.D.
2400 North 38th Street
Kansas City, Kansas

Recent Trends in Therapy of Carcinoma of the Breast

Tumor Conference

Edited by Bernard Klionsky, M.D., and Chauncey G. Bly, M.D.

Dr. Stowell: We are fortunate to have a number of specialists and authorities visiting our tumor conference today. These men are Coordinators of Cancer Teaching in their respective medical schools and have been invited to participate in today's discussion.

We have two cases today which emphasize certain current approaches to the problems in therapy of breast cancer. Dr. McClure, would you tell us about the first patient, please?

Dr. McClure: This patient (Case No. 53-80) is a 56-year-old white unmarried woman. She dates the onset of her present illness to an episode six years ago, when she was struck in the chest by a box full of shoes. She had no symptoms, however, until one year ago, when she first noted the appearance of a mass in the midline just over the xiphoid process. This mass was tender from the outset and gradually increased in size. She was informed by a number of physicians and osteopaths that it was probably nothing serious. One of them was said to have performed some sort of tests on her blood and to have then informed her that it was a tumor but not a cancer. She was first seen here after the mass had continued to enlarge and become more and more painful.

Her past history and family history are noncontributory. Physical examination is entirely negative except that she has this 6 cm. mass over the xiphoid process on the chest. This mass is firm, erythematous, and somewhat painful to touch. It is fixed to the underlying tissue structures. There are no palpable masses in either breast or in either axilla. She is in the hospital at present for diagnosis and treatment as indicated.

Dr. Stowell: Dr. Miles, would you like to comment about this patient or about the diagnoses which your surgical group has been considering?

Dr. Miles: We are not certain yet that this is a breast tumor. The mass is certainly not that of the typical breast carcinoma. We have given serious consideration to the possibility that this mass is a sarcoma arising from the midline chest wall. Our plans are to biopsy this mass first for pathologic diagnosis. If this is proved to be mammary carcinoma, it is in a location where the ordinary surgical approach is unsatisfactory. We may then proceed with a partial resection of the chest wall or with a radical mastectomy and removal of the sternum.

Dr. Stowell: Dr. Nathanson,* would you comment relative to this case?

Dr. Nathanson: Have x-rays of her skeletal system been made?

Dr. McClure: She has had many recent x-rays of her chest, all of which are normal.

Dr. Nathanson: Naturally, I would like to have examined the patient more thoroughly than time permitted. My first impression on gross inspection is that this is a carcinoma arising in the medial portion of peripheral breast tissue. However, I agree that some manifestations are suggestive of a sarcoma. Certainly in considering curative surgical therapy for this patient one would have to entertain the possibility of resecting the internal mammary chain. This tumor is located in an area where spread often includes direct invasion or involvement of the internal mammary and other mediastinal lymph nodes.

I would be interested in what Dr. Newell† has to say concerning the possible value of radiation therapy in this case.

Dr. Newell: We too would wait until a diagnosis had been made on a biopsy specimen. Let us assume that the diagnosis proves to be adenocarcinoma of the breast. If there are metastases within the chest, with all due respect for Dr. Nathanson's skill and hopefulness, I am skeptical that we could find a patient with such spread of the cancer who could be cured by removing the primary tumor and the metastases in the internal mammary chain of nodes. In our experience, one cannot expect to cure a patient by operation if mammary cancer has metastasized to supraclavicular nodes. Do you know of any patients who have been cured by surgery after the cancer has spread from the breast into the supraclavicular nodes?

Dr. Nathanson: Yes, I know of such cases, but have never personally seen any. Halsted[1] in his original description claimed apparent control of the tumor after supraclavicular resection in patients with involved lymph nodes in this area. At the time,

Cancer teaching activities at the University of Kansas Medical Center are aided by grants from the National Cancer Institute and the Kansas Division of the American Cancer Society. Dr. Klionsky is a Trainee of the National Cancer Institute.

* Dr. Ira T. Nathanson, Department of Surgery, Harvard Medical School, Boston. Deceased May 3, 1954.

† Dr. R. R. Newell, Department of Radiobiology, Stanford University School of Medicine, San Francisco.

this revived interest on our part, but our results continued to be poor. I think you may have misunderstood me about the possibility of curing a patient by resection of the internal mammary chain of lymph nodes. I don't entertain any great hope for this procedure, but I think it is feasible to try it.

Dr. Newell: I am enthusiastic in supporting surgical investigation, and I don't think that these radical procedures should be abandoned. I'm just predicting that surgery will not cure the patient in such a case. Neither will radiation produce very many cures, if any, where there are deep lymph node metastases, although it may arrest the tumor for many years in some cases.

We are fortunate that in talking about cancer of the breast we have, as a group, seen enough patients so that we can talk intelligently about the percentage chances of a given outcome in a given patient. I like very much the objective approach which Dr. Haagensen uses. Probably all of you who have had anything to do with cancer are acquainted with his magnificent monograph on the diagnosis and treatment of cancer of the breast.[2] His study appeals to me particularly because, in each case returning with recurrence of cancer, he has carefully studied the complete situation to discover which circumstances or signs would be important in the prediction of recurrence. In his Table 1, he lists eight groups of cases of cancer of the breast in which surgery did not achieve a single permanent cure. He concluded that these eight groups of cases with breast cancer were categorically inoperable, i.e., radical mastectomy would be futile:

1. Extensive edema of skin over the breast
2. Inflammatory carcinoma of the breast
3. Carcinoma developing during pregnancy or lactation
4. Satellite nodules in skin over the breast
5. Intercostal or parasternal nodules
6. Edema of the arm
7. Proved supraclavicular metastases
8. Distant metastases

We have seen a few exceptional survivals in some of our own cases which would belong in the first group of cases. We might consider the problem of trying to place today's first patient into one of the above categories on the basis of superficial gross examination, assuming that she has breast cancer. Biopsy and further exploration may produce findings which will place her in group 1, 5, or 8. Or she may now fit into Haagensen's ninth category, where the patient shows two or more of the five following signs of locally advanced carcinoma: skin ulceration, limited skin edema, solid fixation to chest wall, large (2.5 cm.) axillary nodes containing metastatic cancer proved by biopsy, fixation of axillary nodes to skin or deep tissues.

I was surprised to find the skin not fixed over the breast tumor in this patient. I would have expected to find it, but it cannot be demonstrated. There is fixation to deep tissue structures. She does not have palpable metastases in the axilla or other areas. Accordingly, one might stretch a point and decide that this is perhaps operable, after all, by removing that portion of the chest wall. I would agree with Dr. Nathanson that this might well be a reasonable thing to do. I wish again that we had more clinical investigators who would go through the findings in a purely objective fashion so that we can know which are the important findings by observation of enough cases.[3]

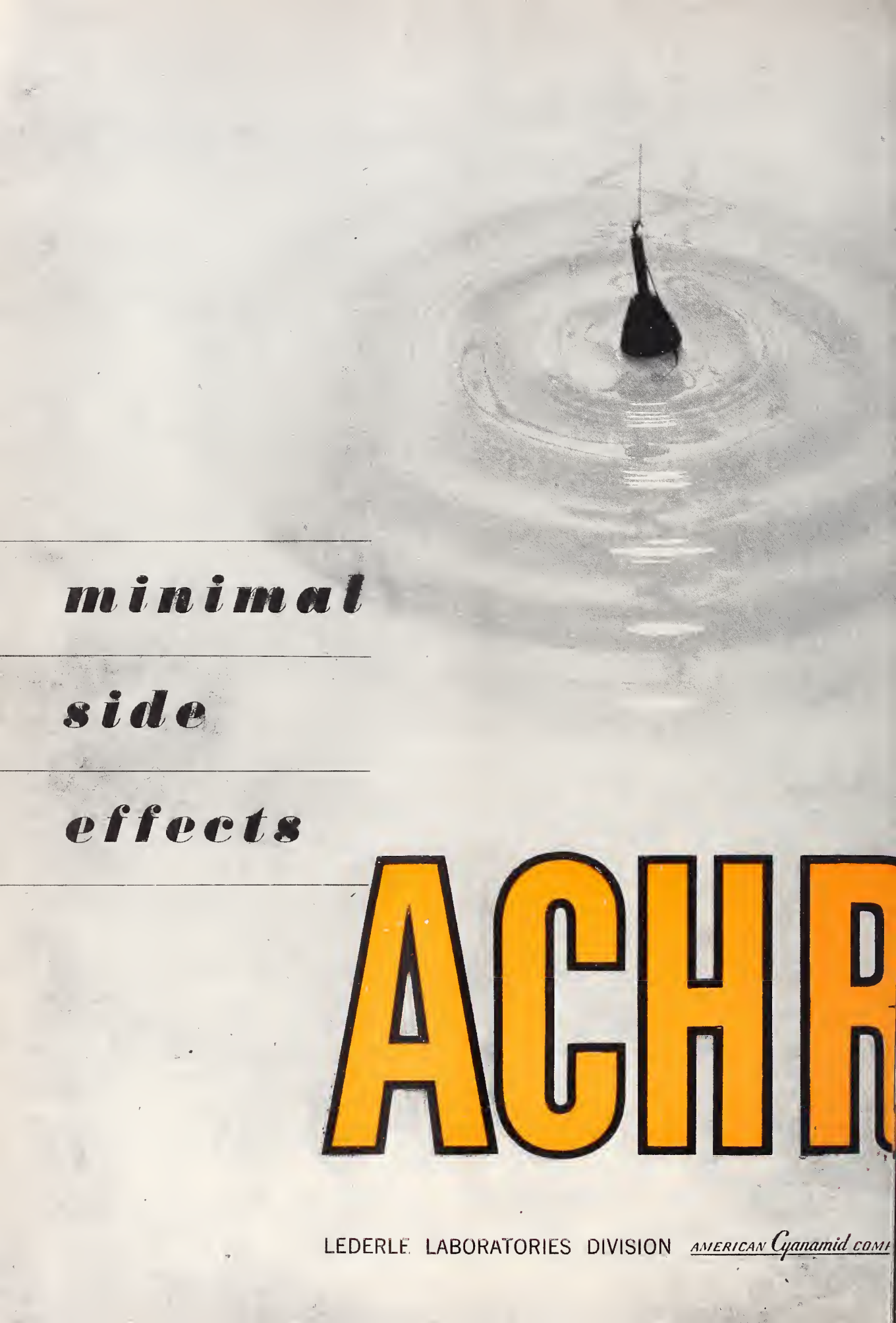
Has anyone else here had personal experience with cases in which there have been metastases to supraclavicular nodes or to mediastinal lymph node chains, and in which there has been a satisfactory period of survival following radical resection of these areas of spread?

Dr. Hitchcock:* I'd like to discuss with you Dr. Wangensteen's rather radical approach to the problem of breast cancer. His idea was that the operation for spreading breast cancer may be extended beyond the standard Halsted type of radical mastectomy to include a dissection of supraclavicular, mediastinal, infraclavicular, subclavicular, mediastinal and internal mammary lymph node chains. In this type of case, he does first a standard Halsted type of radical mastectomy, including removal of the axillary contents. Then he makes a midline incision and splits the sternum. This permits the clavicle to be reflected and the chest wall to be opened to permit access to the entire mediastinal, internal mammary, and supraclavicular areas.

Of the patients with breast cancer who have metastases in axillary nodes, about two-thirds also have involvement of supraclavicular and internal mammary nodes. The internal mammary lymph nodes particularly are frequently involved in those cases where the tumor is placed medially. Deeper and posterior mediastinal structures have rarely been involved. This procedure has been done now on about 60 patients since it was started three and one-half years ago, and it's too early yet to tell what the ultimate data on survival will be.

Personally, I am not altogether satisfied with it yet since the results, in terms of patients permanently free of cancer, are not as good as we would like to see. Only about 30 to 35 per cent of the patients are still clinically cancer-free, three to three and one-half years after the operation. We would prefer

* Dr. Claude R. Hitchcock, Department of Surgery, University of Minnesota Medical School, Minneapolis.


minimal
side
effects
ACHR
LEDERLE LABORATORIES DIVISION
AMERICAN Cyanamid COMP

One of the notable qualities of ACHROMYCIN, the Lederle brand of Tetracycline, is its advantage of minimal side effects. Furthermore, this true broad-spectrum antibiotic is well-tolerated by all age groups.

In each of its various dosage forms, ACHROMYCIN provides more rapid diffusion for prompt control of infection. In solution, it is more soluble and more stable than certain other antibiotics.

ACHROMYCIN has proved effective against a wide variety of infections caused by gram-positive and gram-negative bacteria, rickettsia, and certain virus-like and protozoan organisms.

ACHROMYCIN ranks with the truly great therapeutic agents.

OMYCIN*

HYDROCHLORIDE
Tetracycline HCl Lederle

River, New York

Lederle

*REG. U.S. PAT. OFF



Figure 1. Cords of poorly differentiated adenocarcinoma of the breast (Case 53-80) permeating lymphatics and invading subcutaneous areolar tissue and pectoralis muscle. Hematoxylin and eosin stain. X 92.

to succeed in 70 to 80 per cent of cases. We are continuing to do this procedure in carefully selected patients.

Dr. Nathanson: What are your criteria for selection of patients to receive this operation?

Dr. Hitchcock: For one thing, we don't use it on patients who have known widespread metastases. In considering today's first patient, I would suggest a complete bone survey for metastases. By that I mean x-ray examination of the complete vertebral column, ribs, sternum, pelvis, and upper ends of the femora. I would do all that, as well as a good history and physical examination, in order to rule out, so far as possible, any distant metastases. We might add that the cases which have been selected for mediastinal dissection are either those that have had regional involvement at the time of the original operation or those in whom axillary involvement was subsequently found.

Dr. Nathanson: I would like to support this emphasis on the more objective criteria of selection. So often it is easy for us to argue the points about clinical staging based only on physical examination. Even in some of our clinics of highly trained examiners with long experience and skill in the physical examination of breasts and axillae, the purely clinical staging and prognosis may be variable and fallible. One of our most difficult problems, in deciding what degree and nature of radical surgery will be necessary, is related to the common and typical carcinoma of the breast. This usually shows a primary nodule 2 cm. or so in diameter in the upper outer quadrant and one or two palpable axillary nodes ranging up to 1½ cm. in diameter, a non-specific size which is not necessarily diagnostic of lymph node metastases.

So often patients have lesions of that character, and subsequently in their course one or two nodes become definitely involved. Even so, this group of patients with one or two axillary nodes involved by cancer of the breast may have a high percentage of survival, ranging up as high as 60 per cent at five years after standard radical mastectomy.

Editorial Note: Examination of the biopsy specimen removed from this patient the next day revealed adenocarcinoma with invasion of skin. Three days later, surgical procedures included a right simple mastectomy, left radical mastectomy, removal of the sternum, and dissection of the internal mammary nodes. The carcinoma of the left breast had invaded pectoral muscles and lymphatics (Figure 1), extended to the sternum and to the right breast tissue, and was found in one of the internal mammary nodes examined but in none of the left axillary or left thoracic lymph nodes. Seven months postoperatively, a skin ulcer at the site of mastectomy contained recurrent carcinoma.

Dr. Stowell: Let us move on to the second patient. Dr. Miles will present the history.

Dr. Miles: This woman (Case No. 53-81) was 37 years old when first seen in our clinic three months ago. During the third month of her first pregnancy, at the age of 29, she noticed a lump in the upper outer quadrant of her left breast. Following biopsy, a radical mastectomy for mammary carcinoma was done. Although postoperative radiation therapy was given to the chest wall, axilla, and ovaries, the pregnancy continued to an uneventful delivery of a normal baby. The patient had hot flashes following the radiation to the ovaries. No hormones were administered to inhibit lactation, but endocrine therapy with testosterone was begun one year after the mastectomy and was continued for four years. She noted voice changes during the first year of androgen therapy and menstrual periods during the fourth year, before discontinuing therapy.

At this time, five years postoperatively, she developed pain of a nature which suggested skeletal metastases, although such lesions could not be demonstrated radiographically. Surgical castration at that time resulted in alleviation of pain for about one year. Several courses of radiation therapy have been administered during these years, particularly to vertebrae, with relief of pain.

About three months ago she was referred here

for consideration of bilateral adrenalectomy, almost nine years since her radical mastectomy. X-ray films showed compression fractures of the tenth thoracic and the third lumbar vertebrae; a large mass in the base of the right lung was considered to be a metastasis. At that time she was in severe pain. We discussed the contemplated procedure at some length with the patient, explained that it was experimental, and pointed out to her that there had been some good as well as some unsatisfactory results.

Two months ago a bilateral adrenalectomy was done at one stage. After surgery this woman was almost euphoric. We suggested to her that initially perhaps there would be a large psychic element in this feeling of relief and well-being. However, I am glad to say that thus far she is still free of pain, 65 days postoperatively. We had planned further irradiation of her vertebrae, but she is pain-free at present so we will reserve that for the future. We are sure that she will need it later on. She is my third patient who has had complete relief of pain following bilateral adrenalectomy. One has been pain free for over six months.

We are presenting her case for several reasons. Most unusual is the length of her survival with carcinoma of the breast, diagnosed and treated during pregnancy, and with known widespread metastases for several years. She is well adjusted to her situation. She is now taking 50 mg. of cortisone and 2 mg. of desoxycorticosterone daily.

How much pain were you having when you came here, Mrs. B.?

Patient: I was in such pain that I didn't care whether I lived or not. I couldn't take it any more. When it was really bad, I had to have someone help me up and undress me. I had to have pillows propping me up all over, and it was still terrible. Empirin and codeine pills didn't have any effect on the pain, even for an hour.

Dr. Tice: Have you coughed up any bloody sputum at any time?

Patient: Yes, about six or seven weeks ago in the hospital, I was sleeping on my side, woke up coughing, and spit up a little blood for the first time. It hasn't happened again since then.

Dr. Stowell: Dr. Helwig, would you tell us the significant pathological findings in the specimens removed at surgery? I believe also that Dr. Tice has some pertinent films illustrating some of the lesions.

Dr. Helwig: This was a classical adenocarcinoma of the breast which had metastasized to both adrenals (Figure 2) and to a sympathetic ganglion near one adrenal. Several nodules of tumor up to 2 cm. in diameter are present in these tissues.

Dr. Tice: These films, taken elsewhere, show a normal-appearing chest seven years ago, but radiolucent areas in many of the bones, indicating widespread skeletal metastases two years ago. Our film of her chest taken two months ago shows this 2-3 cm. mass in the basilar portion of the right lung. She may well expect to cough up occasional blood-tinged sputum again in the future. Metastatic involvement seems to be particularly prominent here in the lumbar spine and dorsal spine.

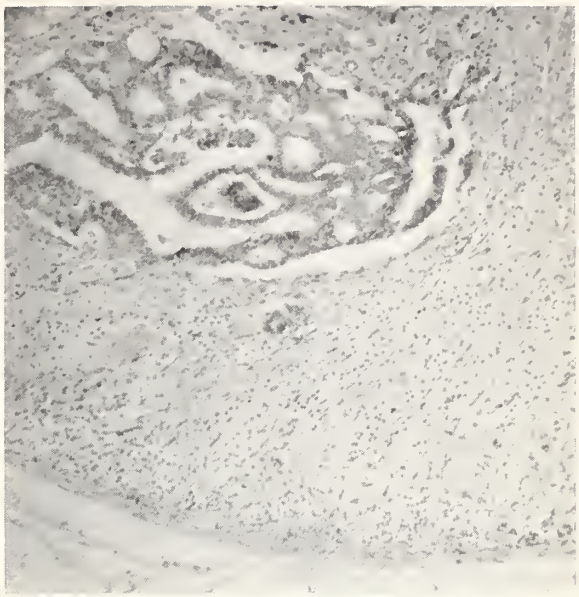


Figure 2. One of many nodules in the adrenals, showing fairly well-differentiated adenocarcinoma from the breast (Case 53-81). Hematoxylin and eosin. X 92.

Dr. Stowell: There are many interesting facets of this case. I'd like to ask Dr. Nathanson if he would again open the discussion, please.

Dr. Nathanson: As Dr. Miles has said, this is an unusual clinical course for a patient with carcinoma of the breast beginning during pregnancy. Pregnancy *per se* is apparently no longer a contraindication to radical mastectomy. We have several cases, operated on during pregnancy, who have lived five years. I'm sure that many others of you here know of a few patients who have also survived for five years.

We've been interested in methods of castration for many years. The technique of radiation castration has survived, to a certain extent, in our own institution, although we were very unhapy with it in the 1930's. At that time, many patients who had received radiation to the ovaries initially had hot flushes and changes in the vaginal cytology characteristic of castration, but in time the menses returned. Studies of the amounts of estrogenic hormone excreted in the urine by these patients revealed that they were not the same as surgical castrates because they had fluctuations of estrogens

in the urine. Indeed, by following the urines, one could predict a resumption of menstruation in a number of these patients. The order of magnitude of radiation dose given in those days was calculated to be approximately 400 to 600 r delivered to the ovaries. It was harder to cause cessation of ovarian function in young women and in fat women, for obvious reasons.

We therefore began to re-evaluate surgical castration which had been performed commonly in the early 1900's but had fallen into disrepute because the results were not consistent and because there was a high morbidity and a certain mortality. No one had re-explored the procedure with any degree of consistency under the regimen of modern surgical technique. At the same time, we ran two parallel series, using surgery in one and giving larger doses of radiation of the order of magnitude of 1,500 and 2,000 r delivered to the ovary in the other. We have seen no resumption of menstruation in either series.

Whether surgical castration or a large dose of radiation is the better method of ablating ovarian function, I am not prepared to say. There is some good evidence, however, from work done in our laboratory by Dr. Engel and others, that it may be beneficial to retain some part of the ovarian function. For example, the stromal apparatus may secrete a hormone, unidentifiable as yet but suspected by many workers, which may be inhibitory to breast cancer. I say this because we have been impressed clinically by apparent acceleration of breast cancers in a number of cases following surgical castration. This is paradoxical because usually we have noted obvious regression of breast cancer following castration.

Very obviously, this present patient did not have complete cessation of ovarian function after radiation, and that is why surgical castration was carried out. The patient therefore showed a satisfactory response to castration with relief of pain for about a year. She did not have demonstrable skeletal metastases, but they were suspected. The tumor was leisurely in its course, unusual in a woman of this age.

I would not want to make any rules about response to hormonal methods of therapy, but the slow-growing tumor that recurs late is usually responsive. The effect of testosterone was hard to evaluate in this patient since we don't know the dosage levels. I certainly would have strongly favored doing an adrenalectomy in this case where every other course had been exhaustively tried. However, I think that one still has to regard adrenalectomy with a certain amount of conservatism. One cannot so treat every patient with signs of recurrence. As you have noted in this patient's course, much more conservative measures succeeded in alleviating her symptoms and signs for a long period of time, and in many other individuals we see the same thing. Only when all other chemotherapeutic measures have been exhausted, and the tumor has adapted itself to each new environment, would I consider the more serious procedure of adrenalectomy. This permanent removal of adrenal tissue imposes upon the physician serious medical and social responsibilities for the care of the patient, for that patient is entirely and continuously dependent upon medical guidance for the maintenance of her life.

In our own hospital we have performed about 15 bilateral adrenalectomies. The main purpose was to see what we could find out about cancer of the breast. These patients have all been studied thoroughly in the laboratories. The point that Dr. Miles made about pain is one that is obvious and gratifying to everyone who has tried adrenalectomies as a last resort. The interesting relief of pain, which comes quickly after the operative procedure, is much like that seen following orchiectomy in cases with prostatic cancer and pain. Patients cannot be talked out of it because they really do feel better.

The question has arisen, however, as to whether the euphoria and relief of pain are really due to adrenalectomy or to the administration of cortisone, particularly in the postadrenalectomy stage. At the level of 50 mg. of cortisone which this patient is now receiving each day, one does not expect to see much euphoria, although it may occur. We have recently revived our interest in the use of cortisone itself as definitive therapy in patients with advanced breast cancer. We are now using much larger doses than we did several years ago when the material was expensive and not readily available in large quantities. We, as well as others,[4] have been able to produce euphoria and relief of pain in some people by the use of cortisone alone. This constitutes a partial "medical adrenalectomy," secondary to the suppression of pituitary trophic factors such as adrenocorticotropin (ACTH). Although I believe the term "medical adrenalectomy" is poor, I think this is an important procedure to consider.

In Stockholm a neurosurgical group under Dr. Herbert Olivecrone has recently performed about 30 hypophysectomies in patients with breast cancer. They have noted favorable responses in about one-third of the patients. However, they have not evaluated such important effects as suppression of ovarian and adrenal functions by the hypophysectomy. These procedures are highly experimental and have not been used long enough to evaluate fully. They are palliative, and I do not think any of them

will provide the answer to the permanent control of mammary cancer. I personally believe that any shift in hormone balance, however produced, may result in regression of a hormone-dependent tumor, although the tumor may never be completely destroyed.[5]

I will cite the course of one patient that I think might be a case in point. In 1949, testosterone produced a striking response in a patient with extensive skeletal metastases. She had previously been bedridden, but within a matter of a few months she was able to work in her garden. In 1950 she returned to the clinic with large masses and radiolucent areas in her skull and tissue masses in the preauricular and peristernal areas. All treatment was stopped. In six months, all of the known lesions regressed, without any further treatment. Had we done an adrenalectomy, or had we given estrogen therapy at that time, we would have attributed the favorable response to the specific therapy.

In July of 1951, her liver, which had always been palpable, suddenly became markedly enlarged and nodular. However, we did nothing about it until September, when her liver filled the entire abdomen, and one could see the irregular masses bulging beneath the abdominal wall. She was markedly icteric and bedridden. At this time she was given ethylene estradiol. The jaundice disappeared and the liver shrank to about half its size. This patient was later being considered for adrenalectomy, but her cardiac status precluded any operative attempt, and she actually died of cardiac disease.

Thus we have observed, in one patient, a shift of endocrine status three times, with regression of tumor each time. Cessation of hormonal therapy to which a tumor has become refractory is not uncommonly followed by regression of the tumor. One may expect any impact on the hormonal balance to produce another respite, although the first shift is usually the most successful.

Dr. Helwig: There are two questions I would like to ask Dr. Nathanson. First, in that group of patients in whom apparently inadequate irradiation castration was followed by surgical castration, what evidence was there of stromal hyperplasia in the ovaries? Second, would you mind commenting on the work by Dr. Engel and yourself on the metabolic transformation of androgens to estrogens? We've often surmised that that's what happened, but you're the first to prove it.

Dr. Nathanson: I'm sorry that I can't answer that first question. Our work was done many years ago. We were not thinking about stromal hyperplasia at that time, and no one has yet gone back over the material.

We really aren't sure yet about the conversion, either. I was interested in treating breast cancer with testosterone back in the 1930's. We wanted to study the steriod secretion pattern in patients with cancer and other diseases. To the group of castrated females, some with cancer of the breast and some with other lesions, we administered testosterone and studied their excretions of gonadotropins, estrogens, and 17-ketosteroids. Much to our amazement and interest, we found a marked increase in biologic activity of estrogenic hormones in the urine of these patients receiving testosterone. We were also able to show in some patients a cornification of the vaginal epithelium indicating estrogen activity which was not present previously. We ourselves are somewhat skeptical of these results because of the inadequacy of the methods used at that time. However, Dr. Louis Engel has recently developed an accurate technique for the estimation of urinary estrogens and can identify the estrogens.

There is a real excretion of estrogens after androgen administration. Whether this is due to conversion or to stimulation of estrogen formation, I don't know. It has long been known of course that stallion urine and testes contained large amounts of estrogenic material. We are inclined to believe at present that there is good evidence for conversion, and we are trying to prove this with tracer experiments using radioactive steroid hormones.

Dr. Newell: As Dr. Nathanson has aptly pointed out, response to hormone therapy does not yield to any logical analysis. But in these already hopeless cases, whenever the cancer escapes from previous control, I too am willing to make another change—stop everything for a while, or start another hormone.

For years we gave routinely 600 r or 800 r to the ovaries as a castrating dose for breast cancer in women before the menopause, except in those patients with Stage I cancer. Lately we have felt little confidence in the effectiveness of this castration, in spite of the fact that we have had a couple of nearly miraculous arrests of tumor growth after irradiation for metastases to pelvic bones in young women. Halberstaedter[6] writes that castration was not effective in cases of breast cancer with metastases in liver or lymph nodes, but that half of his cases in Stages II and III were benefited. However, the natural irregularities of the course in breast cancer make the statistical analysis of data uncertain and precarious.

I have no question about the value of radical mastectomy, in spite of some recent articles to the contrary. I have no doubt at all that there's a big difference in prognosis between those patients who have axillary nodes involved and those who don't. I refer you to the curves of life expectancy in normal

women and in women with breast cancer, shown in Figure 47 by Haagensen.[2] One must again be careful about accepting gross statistics and realize that individual biological variations and fluctuations may make it hard to get statistically significant results. There are times when many of us would rather rest on our intuition, built over years of experience, without submitting everything to statistical analysis before confidently going ahead.

The extremely hazardous outlook of the average pregnant or lactating woman who develops breast cancer is well known to all of us. I too would castrate such a patient. And I believe that as soon as indicated one might also give testosterone to the patient so as to push the alteration of her constitution just as far as possible away from the internal environment which was favorable to the development and growth of her cancer.

Dr. Orr: What are we going to do if a patient, such as this second one today who has had about every treatment available, develops recurrences of symptoms after the adrenalectomy? It's doubtful that her tumor will be permanently repressed by this change in body environment either.

Dr. Nathanson: I am afraid that I agree with you. If her tumor merely shifts its hormone-dependency, we may perhaps succeed again in ameliorating symptoms by one or more changes in hormonal environment—it may not make much difference how the change is produced. On the other hand, if her tumor ultimately loses its hormone-dependency and becomes completely autonomous, as some human and animal tumors do, then nothing may change its course. We'll just have to keep trying our best to keep up with the tumor, even though some of the steps have no known logic.

We like to believe that, in addition to providing gratifying relief of pain as well as permitting the common repair of bony defects caused by metastases in these patients, we have also lengthened these patients' lives by each therapy or change in therapy. However, I'd like to point out that Dr. Welch and I[7] indicated that, without any treatment at all, 20 per cent of patients with proved breast cancer lived five years after the first symptoms. We have also recently attempted to calculate the increased survival in almost 1,000 hormone-treated patients with breast cancer. As yet we can't do it satisfactorily; variations in the data are too great for present statistical methods to give a satisfactory difference. We hope someday to demonstrate a figure for increased survival which will pass everyone's statistical requirements. I think in some patients probably it does happen.

Dr. Newell: I don't believe any of us really needs to calculate a statistically significant increase in average longevity. All we need to do is to pass in review the large numbers of patients who have done well under hormone or other therapy and not be upset by those we were unable to benefit by present methods, or by those few who might have seemed to do worse. To me, inability to bring benefit to those unfortunate patients with unresponsive or uncontrollable tumors is of far less weight than the ability to lengthen the useful, comfortable or tolerable portion of life for a patient who does respond.

BIBLIOGRAPHY

1. Halsted, W. S.: The results of operations for the cure of cancer of the breast, Ann. Surg. 20:497, 1894.
2. Haagensen, C. D.: Carcinoma of the breast. Monograph, American Cancer Society, 1950.
3. Shimkin, M. B.; Lucia, E. L.; Low-Beer, B. V. A., and Bell, H. G.: Recurrent cancer of the breast, Cancer 7:29-46, 1954.
4. Segaloff, A.; Carabasi, R.; Horwitt, B. N.; Schlosser, J. V., and Murison, P. J.: Hormonal therapy in cancer of the breast. VI. Effect of ACTH and cortisone on clinical course and hormonal excretion. Cancer 7: 331-333, 1954.
5. Nathanson, I. T.: Clinical investigative experience with steroid hormones in breast cancer, Cancer 5: 754-762, 1952.
6. Halberstaedter, L.; Hochmann, A., and Lorenz, W.: Die Bedeutung der Menopause für das Mamma Karzinom, Strahlentherapie 80:77-84, 1949.
7. Nathanson, I. T., and Welch, C. E.: Life expectancy and incidence of malignant disease: Cancer of the breast. Am. J. Cancer XXVIII:40-53, 1936.

A human being is no healthier than his environment. If he lives in an environment in which infection is rife, his chances of avoiding illness are obviously less than those of persons who live where public health and sanitation are good.—*Frank F. Tallman, M.D., J.A.M.A. May 22, 1954.*

Integrated Courses at K.U.

Successful operation of a program in correlated teaching of pathology with other disciplines at the University of Kansas School of Medicine was reported by Dr. Robert E. Stowell, chairman of the Departments of Pathology and Oncology, in the September issue of the *Journal of Medical Education.*

Sophomore students are given the new approach of relating subject matter so that material on the same problem is presented at nearly the same time during the year. The formation of discussion groups with representatives of 11 departments in attendance has been given credit for the success of the program.

Each four-hour pathology period consists of a lecture-demonstration, laboratory session, and discussion. Material not adequately presented in textbooks is covered, ideas are presented to invoke student interest, and orientation is given in the student's reading and laboratory study.

BLUE SHIELD

October 25 is the tentative date set for moving into the new Blue Cross-Blue Shield building at Twelfth and Topeka Boulevard, Topeka. This new building is a result of action taken by the board of directors of Blue Shield and Blue Cross some months ago as a part of the continuous long range planning.

The decision by the trustees of Blue Shield and Blue Cross for the building program was based on the following facts:

1. Present space is inadequate and no further expansion is possible in this location.
2. Rental space elsewhere, which would be sufficient for present and future needs, was not available.
3. Rent is already a sizable item in operating expenses.
4. The new building will stabilize rent expense at a figure somewhat below what is currently being paid.
5. Just as in soundly financed home ownership, the building cost will be amortized, and eventually a much greater saving will be achieved.
6. The building will enable Blue Cross-Blue Shield to plan space to meet present and future needs.
7. Better planned space means more efficient operation.

The new building is composed of a basement and two floors. Footings and columns are sufficient to allow for two additional stories if and when needed. This structure will provide about 22,000 square feet of floor space at a cost of approximately $2,500 per month as compared to $3,250 per month rent for 14,275 square feet of floor space in the present quarters.

The building is simple in design and construction to provide for the most efficient use of space at a reasonable cost.



The new Blue Cross-Blue Shield office building at 12th and Topeka, Topeka, now almost completed. The building is designed for efficiency and is built along simple lines. The outside structure is of cut limestone and brick.

Having such a building is consistent with the desire to provide adequate service to members and participating physicians at an economical cost.

All participating physicians are cordially invited to visit the new building at any time.

Pediatrics Chair at K.U.

Dr. Daniel C. Darrow, physician research investigator in children's diseases, who went to the Children's Mercy Hospital in Kansas City, Missouri, in July, will also occupy the newly created chair of the University of Kansas School of Medicine named the Mercy Hospital Professor of Pediatrics Chair.

A year ago the Missouri hospital affiliated with the Kansas school for improving undergraduate and postgraduate medical education in diseases of children. Dr. Darrow will have research facilities at his disposal at both institutions.

Dr. Darrow's past investigations have dealt with dehydration, shock, acidosis, alkalosis, salt deprivation, fluid balance, protein deficiencies, and potassium deficiencies in various diseases of children. He has been at the Yale University School of Medicine since 1928, as professor of pediatrics there since 1947.

Surgeons Hold Meeting

A meeting of the Kansas Chapter, American College of Surgeons, was held at the Lamer Hotel, Salina, September 12. Dr. James G. Hughbanks, Independence, was installed as president; Dr. Thomas P. Butcher, Emporia, became president-elect, and Dr. Orville R. Clark, Topeka, was chosen as secretary-treasurer.

Scientific papers were presented by Dr. Frank F. Albritten, Jr., University of Kansas School of Medicine; Dr. A. G. Dietrich, Newton; Dr. Barrett A. Nelson, Manhattan; Dr. C. K. Wier, Wichita; Dr. W. A. Grosjean, Winfield, and Dr. Hughbanks.

Dr. L. S. Nelson, Salina, showed films taken during a South American trip sponsored by the college.

Establishment of a Leukemia Studies Section in the Laboratory of Biology at the Public Health Service's National Cancer Institute was announced recently by Dr. John R. Heller, director. The new section will be responsible for formulating and executing the program of the National Cancer Institute in experimental leukemia. The work will include investigations directed toward improving the treatment of clinical leukemia and elucidating the etiology and pathogenesis of leukemia in experimental animals.

ACTIVITIES OF MEMBERS

Dr. and Mrs. D. V. Conwell, Wichita, went to London in July to attend the Third International Congress on Gerontology, July 19-28. Dr. Conwell presented a paper, "The Use of Psychological Tests in Determining the Prognosis and Treatment Needs in Geriatrics Mental Illnesses," prepared by himself, Dr. C. J. Kurth, and Dr. Paul G. Murphy. Dr. and Mrs. Conwell later toured Europe.

Dr. Richard J. Reece, who served as director of the health department for Butler and Greenwood Counties before going to Harvard for postgraduate work, received a master of public health degree from the Massachusetts institution in June and has announced plans to become resident physician with the Kansas City-Wyandotte County Health Department.

The Dodge City Medical Center announces that Dr. Evan R. Williams has joined its staff as a specialist in surgery and obstetrics. Dr. Williams came to Kansas from Evansville, Indiana, where he was resident physician in surgery at the Welborn Clinic.

Dr. Charles McCoy, formerly of Kingman, is now practicing in Hutchinson and is limiting his work to ophthalmology. He recently completed a year's postgraduate work at the University of Pennsylvania Medical School.

Two former Newton residents, Dr. Richard M. Glover and Dr. Dale G. Griswold, have returned to Newton to practice at the Axtell Clinic. Both were graduated from the University of Kansas School of Medicine in 1953. Dr. Glover served his internship at St. Joseph's Hospital, Kansas City, and Dr. Griswold at St. Mary's and Children's Mercy Hospital, Kansas City.

Dr. William C. Menninger, Topeka, spoke on "Emotional Problems of Executives" at the Seventh Annual Industrial Health Conference held at Houston under the sponsorship of the Houston Chamber of Commerce, September 23-25.

Dr. Robert Stevens and Dr. Mildred J. Stevens attended a meeting of the World Medical Association in Rome last month. While abroad they are touring England, France, Switzerland, and Italy.

Dr. L. E. Strode and Dr. W. S. Swart, Girard, were subjects of a feature story in the September 12 issue of the *Pittsburg Sun*. The story reported that the two physicians together have practiced a total of 109 years.

Dr. H. R. Wagenblast has resigned his position with the Wichita-Sedgwick County Health Department to work for his master's degree in public health at the University of California.

Dr. and Mrs. Leo Haughey, Concordia, are touring Europe and Dr. Haughey attended the International Congress of Obstetrics and Gynecology held in Geneva in August.

Dr. J. O. Osborne, who recently completed his internship at the University of Kansas Medical Center, opened an office in Ness City last month.

Dr. Harold H. Jones, Jr., formerly with the Snyder-Jones Clinic in Winfield, is opening an office in Ponca City this month.

Dr. A. W. Butcher, who has practiced in Wakefield during the past 15 years, is now practicing in McPherson in association with Dr. R. P. Watterson.

Dr. Jack C. Dysart, Sterling, and Dr. E. Raymond Gelvin, Concordia, recently became fellows of the International College of Surgeons at a meeting of the United States and Canadian sections held in Chicago.

Dr. Robert M. Brooker, Topeka, addressed the Junior League in that city last month on the subject of "Cancer of the Lung."

Dr. W. W. Orrison, formerly of Minneola, is now practicing at the Collingwood Clinic in Plains. He succeeds Dr. George Hopson who recently moved to Stockton.

Dr. Spencer N. Chaffee announces the closing of his office in Solomon.

Dr. Ray T. Parmley, Wichita, was recently elected secretary-treasurer of the Southern Society of Anesthesiologists.

Dr. E. R. Hill, Lyons, recently spent a month in Chicago taking a course in cystoscopy at the Cook County Graduate School of Medicine.

A feature story in the *Council Grove Republican* on August 20 paid tribute to Dr. B. E. Miller, Council Grove, and Dr. George E. Brethour, Dwight, who have been practicing in Morris County 48 and 45 years, respectively.

Dr. C. Alexander Hellwig, Halstead, attended a meeting of the International College of Clinical Pathologists in Washington early in September and presented an exhibit on thyroiditis prepared by himself, Dr. V. E. Chesky, and Dr. W. C. Dreese.

Dr. Findley Law, Bird City, has been named Cheyenne County health officer.

COUNTY SOCIETIES

Physicians of a five-county area near Reno County attended a meeting held at Prairie Dunes last month. Speaker of the evening was Dr. Bordon Michaeljohn, head of the Department of Medicine, University of Colorado.

The Wyandotte County Medical Society has joined with the organizations of dentists and pharmacists in the county to form the Allied Professions Athletic Association. The first event scheduled by the new body was a golf tournament held on September 16 at Victory Hills.

Dr. Leland Randles, Fort Scott, was elected president of the Southeast Kansas Medical Society at a meeting held in Parsons on September 8. Two other Fort Scott physicians were also elected to office, Dr. Rollin Nevitt as vice-president and Dr. Pratt Irby as secretary.

A meeting of the Wyandotte County Society was held in Kansas City on September 21. Dr. Creighton A. Hardin spoke on "Arterial Grafting" and Miss June Miller discussed "Problems of Hearing and Speech in Children."

Members of the Shawnee County Society met in Topeka on September 7. Speaker of the evening was Dr. J. B. Brown, St. Louis, who discussed plastic surgery for industrial and farm injuries.

Conference on Alcoholism

A Kansas conference on alcoholism, sponsored by the Kansas State Commission on Alcoholism, will be held at the University of Kansas, Lawrence, December 1 and 2. Physicians are invited to attend and to take part in a special group seminar. There will also be presented material of interest to hospital administrators, clergymen, industrial leaders, law enforcement officers, educators, public health and social welfare personnel, attorneys, probate judges, and lay citizens.

The purpose of the conference is to present information in regard to the nature of alcoholism, the extent of the problem, and its social aspects. The conference will not deal with "wet" or "dry" issues.

Among the featured speakers will be Dr. Seldon D. Bacon, director of the Yale University Center of Alcohol Studies, and Mrs. Marty Mann, executive director of the National Committee on Alcoholism, New York.

Complete information on the conference may be secured from Extension Division, University of Kansas, Lawrence, or the Kansas State Commission on Alcoholism, 315 West Fourth, Topeka.

Exhibit Exposes Quacks

Presenting the facts on so-called "medicine men" who have duped the public into spending thousands of dollars in the past on quack medical cures and devices is the subject of the A.M.A.'s newest exhibit. Titled "The Modern Medicine Show," this exhibit exposes such quack healers and food faddists as Bernard Jensen, Gayelord Hauser, Adolphus Hohensee, the Milford Sanitarium in Kansas, Harry Hoxsey, and others.

The exhibit is available for immediate bookings at state and county medical society meetings, state or county fairs, and other public gatherings. Requests should be directed to the Bureau of Exhibits of the A.M.A.

DEATH NOTICES

David Thompson Nicoll, M.D.

Dr. D. T. Nicoll, 90, an honorary member of the Shawnee County Society, died July 12 at his home in Bronxville, New York. Born in Scotland, Dr. Nicoll came to this country during his youth and in 1903 was graduated from Hering Medical College, Chicago. He practiced in Topeka from 1904 to 1946, when he retired.

The Medical Management of Peripheral Arteriosclerosis

John D. King, M.D.
Wichita, Kansas

Atherosclerosis, with or without diabetes mellitus, is the most frequent of all peripheral vascular diseases.[34] Although not the killing disease that is coronary atherosclerosis, it is painful and disabling and may necessitate amputations. The physician should, therefore, be prepared to treat this disease promptly and vigorously.

Monckeberg's sclerosis, while more prevalent in the peripheral arteries, almost never causes symptomatic occlusion. Peripheral atherosclerosis in the great majority of cases is clinically significant only in the lower extremities. However, atherosclerotic occlusion may occur in the upper extremities and lead to all the signs and symptoms seen in the lower. The following discussion refers mostly to disease in the lower extremities, but the same principles of treatment may be assumed to apply to all the limbs.

Dilatation of arteries already damaged by atherosclerosis is practically impossible. Thus, it must be kept in mind that the primary objective of all methods of treatment is to increase collateral circulation.

The first question that arises in the conservative management of peripheral arteriosclerosis is the degree of rest advisable. It is thought by some that in the absence of trophic changes, ulcers, or gangrene, the extremity should be exercised frequently to the point of mild pain to encourage the collateral blood supply.[56] Active exercise does cause increased blood flow during and immediately after its administration, in large part through direct vasodilating action of metabolites.[53] However, active exercise increases metabolism, perhaps enough to more than offset increased circulation.[46, 52] Walking at a rate of 70 or fewer steps a minute and exercises such as Allen has described[2]—in which the legs are hung over the edge of the bed, and the feet turned in and out, flexed and extended, and the toes spread and closed—may in the absence of gangrene be among the most valuable treatments known.[23]

In the case of gangrene, or with rapidly deepening cyanosis constituting a "pregangrenous" state, immediate bed rest is indicated. Rest pain is also an indication for bed rest.[4] The position of maximum circulation is with the feet about six inches lower than the heart, which can be accomplished by placing blocks under the head of the bed.[34, 52]

Postural, or Buerger's, exercises can be utilized with bed rest. The patient is instructed to elevate the limb to an angle of 45° for one or two minutes, during which time the vessels empty. The leg is then dropped over the side of the bed for an equal period, filling the vessels with blood. Finally the leg is placed in a horizontal position for the same length of time. In this manner the vessels are alternately filled and emptied, and circulation is allegedly stimulated. The timing of the exercises is arbitrary and can be determined by the degree of blanching and flushing which takes place.

The value of these exercises is controversial. Edwards feels that postural exercises done for 30 to 40 minutes (five to six cycles) three or four times a day are of some value over a long period of time.[16] Wright[56] and Samuels[45, 46] also advocate their use. Postural exercises have been declared useless by others on both theoretical and clinical grounds.[52, 53] Even if these exercises do actually increase blood flow, they would seem justifiable as a measure to prevent venous thrombosis to which immobilization predisposes.

Several types of baths may be used in the treatment of peripheral arteriosclerosis. In the absence of skin lesions, a modified sitz bath may be employed. The patient sits in about 12 inches of water at 94-96° F. for at least 20 minutes a day. The collateral vessels from the trunk and femoral arteries must be covered by the water in order for the bath to be effective.[56]

The warning is often made that heat should be applied directly to the limb with caution, since heat increases metabolism and may thus worsen ischemia. The safety with which direct heat can be applied is determined by a caudal or lumbar sympathetic block, which should produce a rise in skin temperature of 4° F. or more in order for direct heat to be safe.[34] If direct heat is applied at less than normal body temperature, however, damage to the tissues is unlikely, and this sympathetic block test would seem superfluous whenever the heat can be precisely controlled.

Contrast baths are the immersion of the legs alternately in warm and cold water at intervals of

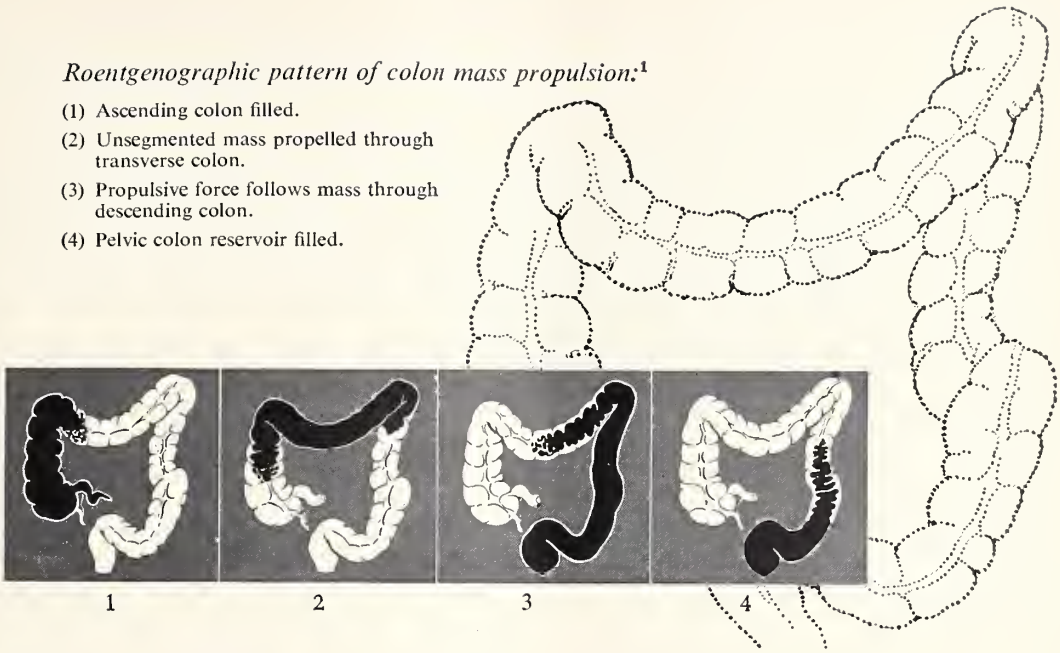
---

This is one of 11 theses, written by fourth year students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group judged to be the best by the faculty at the school.

Thesis written while the author was a fourth year student. Dr. King is now interning at Wesley Hospital, Wichita, Kansas.

Roentgenographic pattern of colon mass propulsion:[1]

(1) Ascending colon filled.
(2) Unsegmented mass propelled through transverse colon.
(3) Propulsive force follows mass through descending colon.
(4) Pelvic colon reservoir filled.



Reestablishing Bowel Reflexes with Metamucil®

Nervous fatigue, tension, injudicious diet, failure to establish regularity, too little exercise, excessive use of cathartics—all factors which contribute to constipation.[2]

Sufficient bulk and sufficient fluid form the basic rationale of treatment of constipation with Metamucil.

Metamucil (the mucilloid of Plantago ovata) produces a bland, smooth bulk when mixed with the intestinal contents. This bulk, through its mass alone, stimulates the peristaltic reflex and thus initiates the desire to evacuate, even in patients in whom postoperative hesitancy exists.

Factors Contributing to Chronic Constipation

Such gentle stimulation is of distinct advantage in reeducating and reestablishing those reflexes which control bowel evacuation. Many factors may pervert the normal reflexes, causing finally chronic constipation. Among them are: nervous fatigue and tension, improper intake of fluid, improper dietary habits, failure to respond to the call to stool, lack of physical exercise and abuse of the intestinal tract through excessive use of laxatives.[2]

Correction of constipation logically, therefore, lies in the suitable adjustment of these factors. The characteristics of Metamucil permit the correction of most of these factors: it provides bulk; it demands adequate intake of fluids (one glass with Metamucil powder, one glass after each dose); it increases the physiologic demand to evacuate; and it does not establish a laxative "habit." Metamucil, in addition, is inert, and also nonirritating and nonallergenic.

Dosage Considerations

The average adult dose is one rounded teaspoonful of Metamucil powder in a glass of cool water, milk or fruit juice, followed by an additional glass of fluid if indicated.

Metamucil is the highly refined mucilloid of Plantago ovata (50%), a seed of the psyllium group, combined with dextrose (50%) as a dispersing agent. It is supplied in containers of 4, 8 and 16 ounces. Metamucil is accepted by the Council on Pharmacy and Chemistry of the American Medical Association. G. D. Searle & Co., Research in the Service of Medicine.

---

1. Best, C. H., and Taylor, N. B.: The Physiological Basis of Medical Practice: A Text in Applied Physiology, ed. 5, Baltimore, The Williams & Wilkins Company, 1950, pp. 579-583.

2. Bargen, J. A.: A Method of Improving Function of the Bowel, Gastroenterology *13*:275 (Oct.) 1949.

one to three minutes. Contrast baths reach only the knee, which is often below the level of occlusion. Furthermore, cold water may precipitate painful vasospasm. The contrast bath is not now used as widely as it once was.[56] Whirlpool baths at about 95° F. may be used to clean away crusts in cases of low grade ulceration or early gangrene.

Thermostatically controlled heat cradles are a satisfactory method of protecting the arteriosclerotic limb from cool drafts and trauma. Maintenance of a uniform warm temperature is important in preventing the cold pressor reflex, which can decrease collateral circulation.[7, 56] Heat should emanate from some sort of conductive heat device, rather than a heat lamp or light bulb, since these are likely to cause burns without close attention. Temperature should be maintained at 92-96° F. except in the occasional case in which pain will necessitate a temperature of 84-88° F.[56] Homemade devices, such as a thermometer placed equidistant from the limb and from a light bulb, require vigilant observation to avoid overheating and are to be condemned.

Woolen socks may be worn to help keep the feet warm. The leg should be propped gently in position with blankets and a folded towel or blanket placed under the Achilles tendon to prevent pressure on the heel which might lead to ulceration.

Shortwave diathermy is a controversial matter. Samuels[46] and deTakats[13] advocate its use, but Wright contends that overheating with attendant thermal injury is too great a danger because of lack of precise temperature control.[56]

Reflex heat refers to application of heating pads to the abdomen, sacral area, or base of the limb, with resulting vasodilatation in the collateral vessels. Heat should not be applied to the extremity itself. Reflex heat is one of the most valuable and effective methods of treatment of peripheral arteriosclerosis.[23, 24]

Avoidance of breaks in the skin is important because healing is slow. Infection is almost certain to occur and difficult to eradicate. Since dermatophytosis is likely to initiate small ulcers and bacterial infection, the prophylactic use of fungicides such as Desenex® and Sopronol® on the feet is advisable.[46] In the absence of open lesions, the skin should be kept clean, warm, and dry.

If ulceration or dry gangrene should develop, soaks of saline, boric acid, or 1:500 azochloramide in tricetin can be used to combat infection. Every two or three days a 1:5000 potassium permanganate soak can be substituted.[56] For moist gangrene or other serious infections, soaks containing 0.5 mg. tyrothricin per cc. or 500 to 5,000 units penicillin per cc. should be applied.[16, 56] Sulfonamide powders should not be used topically, because they tend to crust over and act as an irritant.[56] Soaks of strong antiseptics should never be applied.[55, 56]

It is important to keep soaks warm; cool soaks will chill the leg and cause vasoconstriction. If fever develops, penicillin should be administered intramuscularly. Bacterial cultures and sensitivity tests should be run if possible, to better direct both topical and systemic antibiotic treatment. Infection in the arteriosclerotic extremity is difficult to eradicate because even large systemic doses of antibiotics cannot reach the ischemic tissues in sufficient quantity. Consequently, the first principle of treating infection is a vigorous effort to improve blood supply to the part.

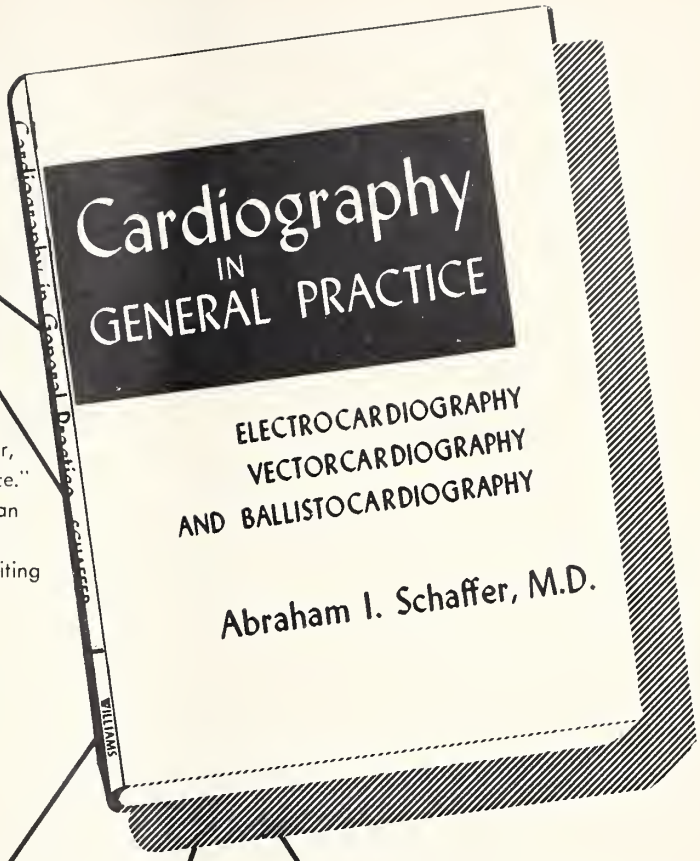
Pressure-suction boot treatment (as with the pavex apparatus) is based on the hypothesis that blood flow to an extremity can be increased by exposing the extremity to alternating positive and negative pressure. The extremity is put into a glass or metal boot, and pressure changes are made by connection through an air pump system. The opening through which the leg is inserted must be made airtight by a rubber cuff, and this cuff may be so tight as to compromise circulation.[56] Although pressure-suction treatment was used for several years it has now generally fallen into disfavor.[31, 36] It is definitely contraindicated in infections, thrombophlebitis, and gangrene.[16, 45]

Intermittent venous hyperemia refers to production of hyperemia following application of a tourniquet for 15 to 20 minutes. Collens and Wilensky reported good results with an apparatus to produce venous stasis and release in cycles of about two minutes by means of a blood pressure cuff around the limb, alternately inflated and deflated by a motorized pump.[10] Many authorities now feel that intermittent venous hyperemia is worthless in the treatment of peripheral arteriosclerosis.[3, 13, 16, 45]

Sanders has devised a "vasoscillating" bed, in which the head and feet of the patient are alternately elevated and lowered, a complete cycle taking one to one and a half minutes.[44] Most patients soon become accustomed to the motion and are able to sleep on such beds for many years. The process is gentle, apparently physiologically sound, and has the additional advantage of reducing the tendency toward thrombosis. It may also reduce the tendency toward demineralization which often accompanies bed rest.[56] A thermostatically controlled cradle may be used as an adjunct. The rocking bed is usually an effective form of treatment, but of course it is expensive and the treatment must be carried on for a protracted period.[34]

Tobacco is widely recognized as being deleterious to an already impaired circulation by causing spasm of the collateral arterioles. Smoking may cause

A repeat offer so that you too may get your **FREE COPY**



This $3.00 book just off the press and yours for the asking. Abraham I. Schoffer, M.D., "Cardiography in General Practice." This book is so important to any physician interested in **EKG**, that we, the mokers of **CARDIOTRON** the original direct writing electrocardiograph, repeat our offer, and want every general proctitioner to add it to his library at no cost or obligation.

Here at last, is a concise, simply written book, dealing with all of the clinical **EKG** problems you face daily in your practice. It will be most invaluable to you.

We know that you will also want to see, at the same time, the most advanced electrocardiograph ever made, the **NEW CARDIOTRON Model PC-3.**

We have bought a limited supply of this important book, so be sure to send for your **free** copy today.

Mail this coupon today

United Medical Equipment Co.
2010 Broadway, Kansas City, Mo.

Please deliver my FREE copy of "Cardiography in General Practice" and arrange to show me the NEW CARDIOTRON PC-3.

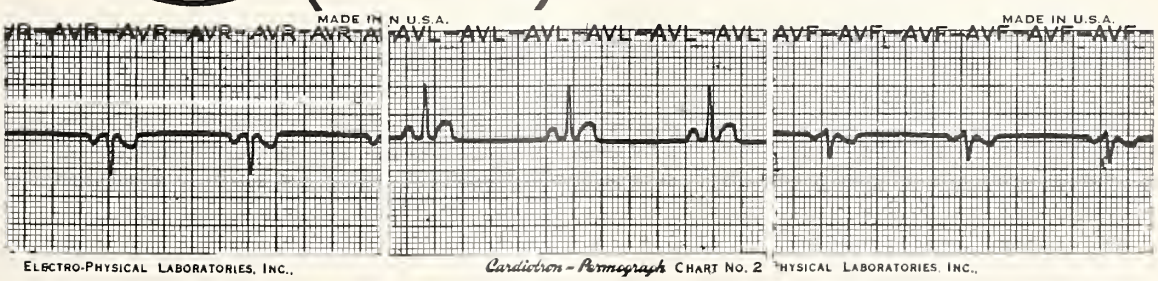
Name.....

Address.....

City.....Zone.....State.....

NOW— For the First Time— PRINTED LEADS— AUTOMATICALLY

One of the many features of THE NEW **CARDIOTRON**



gangrene where circulation to an extremity is already seriously reduced by arteriosclerosis.[23, 55] Lampson has reported that when the smoke of one cigarette is inhaled, vasoconstriction lasts 60 minutes, and that even without inhalation there is 15 minutes of vasoconstriction. Cigar and pipe smoking also induce vasoconstriction.[33] Wright believes that the number of cigarettes smoked has little clinical significance and that complete abstinence is thus indicated.[55] Smoking is a difficult habit to break, however, and may not be a serious danger to the patient with only intermittent claudication.[51] Wright found that standard, denicotinized, and mentholated cigarettes all seem to have about the same effect,[54] but further studies on denicotinized cigarettes have been suggested.[51] In any event, the patient should be told that complete cessation of smoking is desirable.

Anticoagulants, e.g. heparin and dicumarol, have been used to prevent the formation of thrombi. However, they probably are of no practical value in gradually progressing vascular occlusion.[13] In older patients, particularly those with hypertension, the danger of intracranial hemorrhage makes the use of anticoagulants inadvisable.[16] Anticoagulants are definitely contraindicated when surgery is anticipated, when there is an open wound, or if there has been a recent cerebral hemorrhage.[16] Anticoagulants should be used when thrombosis is known to be present. They will not dissolve existing thrombi but do prevent their extension.

When dicumarol is employed, daily Quick prothrombin levels should be obtained. With an initial prothrombin level of about 100 per cent, a dose of 200 to 300 mg. is given, and up to 200 mg. a day subsequently as needed to maintain the prothrombin level close to 25 per cent. If bleeding occurs, vitamin K should be given intravenously.[16]

Dicumarol is relatively inexpensive and can be given orally, but it does not appreciably affect the clotting mechanism for at least 24 hours. Heparin, although expensive and effective only with parenteral administration, acts within a few minutes and may be given with dicumarol for a day or two until the latter takes effect.[17] Heparin is given intravenously, 100 to 150 mg. at first, then 50 to 100 mg. every four hours, or 100 to 150 mg. twice a day, thereafter. It may also be administered in doses of 200 to 300 mg. subcutaneously in Pitkin's menstruum, from which it is absorbed slowly, but pain and induration at the site of injection are common.[16] Venous clotting time should be obtained when heparin is being given. Protamine should be administered intravenously in case of bleeding.[17] The role of heparin in prevention of atherosclerosis will be discussed later.

It is said that insufficient dosage of anticoagulants, particularly dicumarol, favors rather than inhibits thrombosis.[13]

Fever produced by injection of foreign proteins is of definite value in increasing circulation in arterial occlusion. Foley recommends an initial dose of five million typhoid organisms, with an increase of three million organisms in each of the subsequent doses given every two or three days.[23] This dose will cause a fever of only 1.0 to 1.5 degrees F. and is less than that generally recommended for production of fever. However, larger doses cause chills which produce vasospasm and decrease the coagulation time of the blood.[23, 45]

Numerous vasodilating drugs have been tried in peripheral atherosclerosis. Ethyl alcohol has proved to be one of the most successful vasodilators.[23, 55] Two or three ounces of whisky, for example, causes a rise in temperature in the tips of the extremities of 9 to 12 degrees F.[56] With impending or early gangrene it may be justifiable to keep the patient slightly inebriated with one or two ounces of whisky every four hours, or more frequently, until circulatory improvement occurs. The dose can then be gradually tapered off.[56] Whisky may also control pain more satisfactorily than narcotics.

Diabetes is no contraindication to the use of alcohol, but the caloric content of the alcohol (7 calories per gram) must be included within the diabetic diet. Small amounts of alcoholic beverages one or more times a day may be made part of the long-range therapeutic program.[13, 23, 55] Peptic ulcer and alcoholism are contraindications to the use of alcohol.[56]

Many workers have reported the value of pancreatic tissue extracts in the treatment of intermittent claudication.[6, 31, 45, 56] An antagonism to epinephrine has been demonstrated in animals, but not in man.[56] There is conflicting evidence concerning pancreatic extracts, and a final opinion cannot be given at this time.[31, 46] Depropanex® (deproteinated pancreatic extract) given according to a schedule of 3 cc. intramuscular doses three times a week for several weeks, then twice a week for a total of at least 20 doses, has been helpful in some cases.[56] The drug relieves the pain of intermittent claudication by a direct action on the ischemic muscle rather than by vasodilatation,[6] but rest pain is not relieved.[46]

Histamine is a potent vasodilator, but systemic administration is not feasible because its effects are too generalized.[7, 23] However, injection into the femoral artery produces a concentration of histamine in the leg great enough to cause vasodilatation in the leg but not in the rest of the body. With this technique, 2.75 mg. of histamine in 500 cc. of physiological saline, administered at a rate of 60 drops a



Which filter-tip cigarette is the most effective?

IN continuing and repeated impartial scientific tests, smoke from the new KENT consistently proves to have much less nicotine and tar than smoke from any other filter cigarette—old or new.

The reason is KENT's exclusive Micronite Filter.

This new filter is made of a filtering material so efficient it has been used to purify the air in atomic energy plants of microscopic impurities.

Adapted for use as a cigarette filter, it removes nicotine and tar particles as small as 2/10 of a micron.

And yet KENT's Micronite Filter, which removes a greater percentage of nicotine and tar than any other filter cigarette, lets through the full flavor of KENT's fine tobaccos.

Because so much evidence indicates KENT is the most effective filter-tip cigarette, shouldn't it be the choice of those who want the minimum of nicotine and tar in their cigarette smoke?



Kent with the exclusive Micronite Filter

"KENT" AND "MICRONITE" ARE REGISTERED TRADEMARKS OF P. LORILLARD COMPANY

minute, apparently increases blood flow through muscle. Intra-arterial injection is difficult but successful in many cases.[23] Intra-arterial and intracutaneous histamine can also be used to determine the border of viable tissue in a gangrenous extremity requiring amputation.[13]

Nitrites and nitrates (which release nitrites in the body) are potent vasodilators. They have found wide use in arterial occlusive diseases, but there is a lively controversy over the effectiveness of this group of drugs. Some investigators feel that although the nitrites are undoubtedly vasodilators, their action is too feeble and evanescent to warrant their use.[45, 56] Others think that these drugs are effective and practical, especially for arteriosclerosis of the upper extremities.[23, 31, 47] Sublingual nitroglycerine has been reported to produce good vasodilatation in the extremities for 20 minutes after completion of absorption.[47]

In recent years tetraethylammonium chloride (TEAC, Etamon®) and bromide, ganglionic blocking agents, have been given increasing attention. Tetraethylammonium in intravenous or intramuscular doses of 20 mg. per Kg. body weight will produce practically complete inhibition of vasomotor tone in the hands and feet.[24] Sustained improvement has been observed in some patients with peripheral atherosclerosis after several days administration of 250 to 500 mg. TEAC intravenously.[11] Oral administration is not effective.

Favorable opinion concerning the therapeutic value of TEAC is by no means unanimous. Several workers feel that the only practical use for the drug is as a diagnostic and prognostic measure.[37] That is, if TEAC causes considerable vasodilatation, there is an important component of vasospasm, and vasodilator drugs or sympathectomy are more likely to be successful. Side effects, e.g. numbness and tingling in the extremities, are often so disagreeable as to preclude therapeutic use of the drugs.[23] Tetraethylammonium may cause postural hypotension in hypertensive patients; consequently, after patients have received the drugs, they should remain recumbent for an hour.[47]

Pentamethonium (C5) is a ganglionic blocking agent which has shown promise of being a useful vasodilator. An intravenous dose of 30 to 50 mg. produces vasodilatation within a few minutes which persists for at least an hour.[8] Pentamethonium may also act directly on the vessels, since it produces vasodilatation even on sympathectomized limbs. It is effective only when administered parenterally.[8, 22]

Hexamethonium (C6) is closely related to pentamethonium and has similar pharmacologic actions. Given parenterally in doses of 30 to 40 mg., hexamethonium causes effective collateral vasodilatation in most cases. It has been reported as being more



ARISTOCRAT IN ITS FIELD

Audivox, successor to Western Electric Hearing Aid Division, brings the boon of better hearing to thousands.

These are the Audivox Hearing Aid Dealers who serve you in Kansas. Audivox dealers are chosen for their competence and their interest in your patients' hearing problems.

TOPEKA

The Topeka Hearing Center
824 Quincy Street
Tel: 4-9609

WICHITA

Kansas Hearing Center
223 South Broadway
Tel: 4-8614

KANSAS CITY, MISSOURI

Southwestern Hearing Aid Service
202-12th and Walnut Building
Tel: Harrison 0541

ST. JOSEPH, MISSOURI

Southwestern Hearing Aid Company
216 North Seventh Street
Tel: 4-8515

HASTINGS, NEBRASKA

O. E. Kullberg
110 Forest Boulevard

audivox
TRADE-MARK
SUCCESSOR TO
Western Electric
HEARING AID DIVISION



aristocrat

Only a long tradition of breeding and cross-breeding for beauty, size, and color can produce a flower aristocrat.

Only **audivox** in the hearing aid field can trace an ancestry that includes both Western Electric and Bell Telephone Laboratories. **audivox** lineage springs from the pioneer experiments of Dr. Alexander Graham Bell, which were furthered by the development of the hearing aid at Bell Telephone Laboratories, and in turn, brought to fruition by Western Electric and **audivox** engineers.

Distinctly an aristocrat in its field, **audivox**, successor to Western Electric Hearing Aid Division, brings the boon of better hearing, and its enrichment of living, to thousands. With the magical modern transistor, with scientific hearing measurement and scientific instrument-fitting, serviced by a nationwide network of professionally-skilled dealers, **audivox** moves forward today in a proud tradition.

TO THE DOCTOR: *If you use or need an audiometer* there is in every major city from coast to coast a career Audivox dealer, chosen for his integrity and ability, who will be glad to show you why an Audivox audiometer will serve you best.

New Audivox audiometer 7BD ...variety of accessories available

Alexander Graham Bell

audivox

Successor to *Western Electric* Hearing Aid Division

123 Worcester St., Boston, Mass.

The Aristocrat of Audiometers

effective than 50 mg. of priscoline or 400 mg. of tetraethylammonium intravenously.[19] It is effective immediately, and its action persists for 30 to 120 minutes.[8] Like pentamethonium, hexamethonium often causes postural hypotension in persons with hypertension. Side effects, such as tingling sensations, are often severe and limit the therapeutic usefulness of hexamethonium.[23] Both pentamethonium and hexamethonium deserve further therapeutic trials since they are drugs which appear to have an unusual selectivity for vasodilatation in the lower extremities.[8]

Papaverine is a widely-used muscle relaxant and vasodilator about which there is also a divergence of opinion. Several clinicians have concluded that papaverine is of no appreciable value in the treatment of peripheral arteriosclerosis.[34, 35, 39, 45] Although papaverine causes cutaneous vasodilatation, it possibly leads to vasoconstriction in the muscles and thus aggravates gangrene or intermittent claudication.[23]

Wright has found that intra-arterial papaverine produces striking vasodilatation in cases of sudden occlusion, but he believes the drug is unreliable and not as effective a vasodilator as alcohol or reflex heat in gradually progressing atherosclerosis.[56] The relaxing effects of this and other opiates has long been recognized and is undoubtedly valuable, and there is practically no danger of addiction to papaverine.[47] Papaverine is usually administered intravenously in doses of one-fourth to one grain and often produces satisfactory vasodilatation.[13, 31] Samuels has found divided oral doses of 15 to 20 grams a day effective.[46]

A derivative of papaverine, dioxyline phosphate (paveril phosphate), has shown fairly good results, without troublesome side effects, in mild or moderate arteriosclerosis.[14, 27] The drug is administered in divided oral doses of 0.6 to 1.8 grams a day.[14]

One of the more promising new vasodilators is dibenzyline (SKF 688A), a peripheral adrenergic blocking agent. Given intravenously in doses of 1 mg. per Kg. body weight over a period of an hour, dibenzyline may produce almost complete sympathetic block for from three hours to four days.[23, 38] The effect of dibenzyline may persist for as long as a week, because the drug is taken up by fat deposits and slowly released therefrom.[49] Although dibenzyline is irregularly absorbed from the gastrointestinal tract, oral administration of doses of 1 to 25 mg. per Kg. body weight is said to be practical.[38] The oral effectiveness and long duration of action are unique advantages of dibenzyline. Blood flow to the upper extremities is more improved than that to the lower, and circulation to the skin is more improved than that to the muscles.[49]

Despite its potency, Moser and associates feel that dibenzyline is not as effective as ganglionic blocking agents such as pentamethonium, hexamethonium, and etamon.[38] Severe side effects and the development of tolerance may prove to limit its usefulness.[38, 49]

Dibenamine is a peripheral adrenergic blocking agent closely related to dibenzyline. It should be given in doses of 4 to 6 mg. per Kg. body weight intravenously. Hypotension and side actions such as nausea, drowsiness, and nasal congestion occur in more than half the patients receiving it.[26, 34] Given intravenously, dibenamine will almost completely inhibit sympathetic vasoconstriction for up to 24 hours. Clinical experience with this drug has been varied.[13]

A drug which has received wide use in peripheral vascular diseases is priscoline. Priscoline is a peripherally-acting drug having both sympathomimetic and sympatholytic properties; it is sympatholytic in that it blocks the pressor action of epinephrine, but it has the sympathomimetic action of vasodilatation in the muscles.[1, 43] The improvement of circulation in the muscle, as judged clinically and by clearance of radioactive sodium injected into the gastrocnemius muscle, is usually insignificant as compared to the increased cutaneous blood flow.[17] Blood flow through the muscle may even be decreased by priscoline,[39] but others have found priscoline effective in alleviating intermittent claudication and night cramps.[49]

Priscoline has also been reported as effective in increasing cutaneous blood flow in both the upper and lower extremities.[32, 34, 39] Sustained improvement in peripheral circulation for as long as six weeks after the cessation of oral administration may be seen. Priscoline has not produced uniformly good results, however, being judged unreliable or of questionable value.[13, 23, 24, 43] Although priscoline is a selective digital cutaneous vasodilator, it has been judged less effective than reflex heat in this respect.[43]

Priscoline may be administered either intramuscularly or intravenously in doses of 25 to 50 mg. every two to four hours, or orally in doses of 75 to 150 mg. a day. Parenteral administration is most reliable.[21, 42, 43] Headaches, chilliness, nausea (but rarely vomiting), and diarrhea are sometimes encountered with oral administration, but these side effects can be avoided by starting with a dose of one 25 mg. tablet twice a day, increasing to the full dose in two to four weeks.[32]

Roniacol tartrate is the tartaric acid salt of the alcohol corresponding to nicotinic acid. It has been observed to increase pulse volume, improve skin temperature, and alleviate intermittent claudication and night cramps in peripheral arteriosclerosis.[28, 34, 49]

...in 2 hours or less

filmtab*

Erythrocin® Stearate

(Erythromycin Stearate, Abbott)

disintegrates faster than enteric-coated erythromycin

TISSUE-THIN FILMTAB COATING (marketed only by Abbott) actually starts to dissolve within 30 seconds after administration —makes ERYTHROCIN available for immediate absorption. Tests show that new Stearate form definitely protects ERYTHROCIN from gastric juices.

BECAUSE THERE'S NO DELAY FROM AN ENTERIC COATING, your patient gets high, inhibitory blood levels within 2 hours—instead of 4-6 as before. Peak concentration at 4 hours, with significant levels for 8 hours.

USE FILMTAB ERYTHROCIN STEARATE against the cocci . . . and especially when the organism is resistant to other antibiotics. Low in toxicity—*it's less likely to alter normal intestinal flora than most oral antibiotics.* Conveniently sized (100, 200 mg.) in bottles of 25 and 100. Abbott

*TM for Abbott's film sealed tablets, pat. applied for

410203

The dose is one to four 50 mg. tablets orally three or four times a day.[13]

Intra-arterial aminophylline has been reported to give striking relief of intermittent claudication.[41] A 3 ¾ grain dose of aminophylline is injected into the femoral artery in a two-minute period. The increase in circulation may not take place for 12 hours, but it is often apparent for several days. The action of aminophylline is apparently directed upon the arterioles, since it occurs in sympathectomized limbs. Some patients have had no return of intermittent claudication for a year, and the injections have been given repeatedly without ill effect. However, in view of untoward side effects which sometimes follow too rapid intravenous administration of aminophylline, a slower rate of injection would seem advisable. Intravenous aminophylline produces brief cutaneous vasodilatation but is of no value in arterial occlusive disease.[47]

Regitine has recently been tried as a peripheral vasodilator. It is best given in doses of 0.75 mg. per Kg. body weight intramuscularly. Oral administration is unreliable and likely to cause diarrhea. Regitine, like many other adrenergic blocking agents, has side effects such as tachycardia, flushing, and anxiety, and there is often irritation at the site of injection. Regitine is probably somewhat less effective than priscoline, but evaluation of this drug for peripheral arteriosclerosis must await further clinical trials.[9]

Among the xanthines, theobromine has received the most extensive clinical trial. Most workers today are inclined to think that theobromine is of no practical value in improving collateral circulation.[31, 36, 56] Potassium iodide has long been used in the treatment of arteriosclerosis. Clinical results have been disappointing. Iodides will not improve collateral circulation or cause reversal of atherosclerosis.[47, 56] Histidine and ascorbic acid in combination are now known to be without merit.[50]

Orally administered nicotinic acid has been reported to improve cutaneous circulation in the upper extremities, and intra-arterial niacin may have clinical value, but the drug is not highly regarded in most quarters.[31] Intramuscular ethyl ether has been advocated by Katz for the relief of ischemic pain,[29] but few others have been impressed with its results.[50]

The hydrogenated alkaloids of ergot were once thought of potential value because of their sympatholytic action, but these drugs are best avoided in the treatment of peripheral atherosclerosis.[23] For a time, intravenous hypertonic saline was thought to improve collateral circulation.[45] The consensus now is that this measure is completely worthless. The administration of mecholyl (acetyl-B-methylcholine) by iontophoresis is condemned as being ineffective and likely to result in burns,[45] although it is still used by some.[31, 47] In any event, the special technique and apparatus necessary for iontophoresis limit the application of the procedure.

Acetylcholine, a potent vasodilator, and its derivates have too transient an action to be of clinical value.[7] Protenyl® is a manganese salt of acetylcholine which releases acetylcholine slowly in the body. Although some French workers have waxed enthusiastic over Protenyl, clinical experience in this country indicates that it is not a valuable drug.[23]

Other drugs that have been tried and generally found without merit include vitamin E, methyl testosterone, calcium salts, cytochrome C, thiouracil, and sodium citrate.[51, 56]

One principle of hemodynamics may explain indifferent results obtained with some vasodilators. If the resistances of all portions of the peripheral vascular bed are decreased equally, blood flow through any one part will be little increased.[7, 23] To be effective in treatment of peripheral ischemia, a drug must exert a selective action on the vessels of the extremities or increase cardiac output. Vasodilators having a generalized peripheral action will increase local blood flow to a small extent by shunting some blood from the visceral to the peripheral circulation, but this factor appears to be of minor importance.[7] Nitroglycerine, for example, dilates arterioles about equally over the body surface and is effective chiefly by means of increasing cardiac output.[23]

There is usually a reciprocal vasoconstriction in muscles when skin vessels dilate.[7] Thus, vasodilators that markedly improve cutaneous circulation may actually be deleterious in intermittent claudication and deep gangrene.[12]

The success of vasodilators also depends in part upon the degree of vasospasm that follows the organic occlusion.[13, 31] Vasodilators are generally less effective in peripheral arteriosclerosis than in thromboangiitis obliterans, since in the former there is usually a lesser degree of vasospasm.[4, 56]

Another factor making evaluation of therapy difficult is the increase in collateral circulation that commonly occurs in peripheral atherosclerosis even without treatment. This spontaneous improvement is due to the vasodilating effect of ischemia itself and to the increased difference in arterial pressures above and below the occlusion.[51]

Sympathetic block and surgical procedures are not within the strict purview of this discussion but should be mentioned briefly in order to mark more clearly the limits of medical management. Sympathetic block, preferably by injection of 30 cc. of 1 per cent procaine hydrochloride into the caudal canal, is useful chiefly as a measure for predicting the success of sympathectomy, since its duration of action is too transient for practical treatment.[16, 34]



NEW VICEROY GIVES SMOKERS

20,000 FILTERS

in every Viceroy Tip

Only Viceroy has this new-type filter. Made of a non-mineral cellulose acetate—it gives the greatest filtering action possible without impairing flavor or impeding the flow of smoke.

Smoke is also filtered through Viceroy's king-size length of rich costly tobaccos. Thus, Viceroy smokers get *double the filtering action* . . . for only a penny or two more than brands without filters.



WORLD'S LARGEST-SELLING FILTER TIP CIGARETTE

New King-Size Filter Tip **VICEROY**

ONLY A PENNY OR TWO MORE THAN CIGARETTES WITHOUT FILTERS



The value of sympathectomy in the management of peripheral atherosclerosis is difficult to assess. DeTakats is of the opinion that sympathectomy is often successful, even in cases where other evidence of vasospasm is slight.[13] Other opinion is contrary to this optimistic view of the operation.[23, 34] Sympathectomy seems the best course of action in those cases in which the patient has absolutely ceased smoking, where there is progression of arterial insufficiency in spite of continued conservative treatment, and in those patients in whom a sympathetic block produces good vasodilation.[34, 40] Sympathectomy should be tried in arterial thrombosis, even if the response to sympathetic block is poor.[16]

If deep gangrene, even with osteomyelitis, supervenes, conservative measures may be intensified for the period necessary for the gangrenous area to become clearly demarcated. Amputation will of course be necessary in these cases. In the absence of severe infection, small parts, such as the toes, may undergo spontaneous sloughing.[43, 56] If infection continues to extend despite medical therapy, immediate high amputation is advisable in order to protect the integrity of the tissues at the amputation site.[16, 55] The abandonment of other measures in favor of amputation will seem less dreadful when the dangers of pulmonary embolism and infection and the expense of protracted bed rest and other treatment involved in conservative management are considered.[16]

A discussion of atherosclerosis always raises the question of the influence of dietary and other factors upon fat and cholesterol metabolism. There can be little doubt that the level of cholesterol-containing lipoprotein molecules of the Sf 10-20 range bears a close relationship to the development of atherosclerosis. Dietary restriction of cholesterol, and much more importantly, of total fats, has produced a decrease in these lipoproteins in a large proportion of patients.[5, 15, 30, 48]

Keys advocates a sharp reduction in all dietary fats as an effective measure in halting the progress of atherosclerosis.[5] He would allow an ordinary amount of eggs and milk in cooking, but no butter, cream, or other high-fat foods. Gofman and others would agree that severe atherosclerotic occlusion is an indication for a low-fat diet. Page and Katz,[5] however, feel that such restrictions are not indicated unless the patient is overweight or has had a myocardial infarction.

Lipotrophic drugs have not proved of any benefit in the prevention or treatment of atherosclerosis.[5, 56, 48] Thyroid is indicated for even mild hypothyroidism, since this endocrine has a marked influence on experimental atherosclerosis.[48] Thyroid must be given cautiously to patients with coronary or other heart disease. Relatively rigid control of diabetes is advisable to decrease the tendency both to atherosclerosis and to infection.[51]

Heparin has been shown to reduce the proportion of lipoprotein molecules in the Sf 10-20 range and to inhibit atherogenesis in the rabbit.[48] Heparin may even cause some reversal of atherosclerosis, a contention lent weight by the increased digital blood flow and walking tolerance observed following the administration of 100 mg. of heparin intravenously two or three times a week.[18]

Suffice it to say that many more experimental and clinical observations must yet be made to permit definite conclusions about the etiology and prevention of atherosclerosis. At present, the use of heparin and of dietary fat and cholesterol restrictions would seem most logically based on the willingness of the patient to undergo rather drastic, long-term measures of unproved efficiency.

SUMMARY

Atherosclerosis is the most common peripheral vascular disease and deserves early, vigorous medical treatment. Prevention of infection and improvement of collateral circulation are the primary objectives of management. Physical therapy, vasodilating drugs, and other measures are presented and evaluated. Some hemodynamic principles involved in vasodilatation are discussed. The role of lipoproteins in atherosclerosis and the clinical applications thereof are considered.

BIBLIOGRAPHY

1. Ahlquist, R. P., et al.: The pharmacology of benzyl-imidazoline (Priscol), J. Pharm. & Exper. Ther. 89:271 (Mar.) 1947.
2. Allen, A. W.: Recent advances in the treatment of circulatory disturbances of the extremities, Ann. Surg. 92:931 (Nov.) 1930.
3. Allen, E. V., and McKechnie, R. E., Jr.: Effect of intermittent venous occlusion on circulation of extremities, J. Lab. & Clin. Med. 22:1260 (Sept.) 1937.
4. Allen, E. V.; Barker, N. W., and Hines, E. A., Jr.: Peripheral Vascular Diseases, ed. 1, Philadelphia, W. B. Saunders Co., 1950.
5. Arms, A.: Recent concepts of the pathogenesis of atherosclerosis, J. Am. Geriat. Soc. 1:3 (Mar.) 1953.
6. Barker, N. W.; Brown, G. E., and Roth, G. M.: Effect of tissue extracts on muscle pains, Am. J. M. Sc. 189:36 (Jan.) 1935.
7. Best, C. H., and Taylor, N. B.: The Physiological Basis of Medical Practice, ed. 5, Baltimore, The Williams & Wilkins Co., 1950.
8. Burt, C. C., and Graham, A. J. P.: Pentamethonium and hexamethonium iodide in investigation of peripheral vascular diseases and hypertension, Brit. M. J. 1:455 (Feb. 25) 1950.
9. Clark, C. W., Jr.; Hays, D. R., and Van Itallie, T. B.: Clinical appraisal of a new adrenergic blocking agent: effect of Regitine on digital blood flow in normal subjects and patients with peripheral arterial diseases, Circulation 8:715 (Nov.) 1953.
10. Collens, W. S., and Wilensky, N. D.: Use of intermittent venous compression in treatment of peripheral vascular disease, Am. Heart J. 11:705 (June) 1936.
11. Coller, F. A., et al.: Tetra-ethyl-ammonium as adjunct in treatment of peripheral vascular disease and other painful states, Ann. Surg. 125:729 (June) 1947.
12. De Bakey, M. E.; Burch, G.; Thorpe, R., and Ochsner, A.: The "borrowing-lending" hemodynamic phenomenon (hemometakinesia) and its therapeutic application in peripheral vascular disturbances, Ann. Surg. 126:850 (Dec.) 1947.
13. de Takats, G.: Diagnosis and management of peripheral vascular disease, Med. Clin. N. America 36:1 (Jan.) 1952.
14. Deterling, R. A., Jr.: The use of dioxyline phosphate in peripheral vascular disorders, Angiology 4:397 (Oct.) 1953.
15. Editorial: Biochemistry of atherosclerosis, South. M. J. 46:8 (Aug.) 1953.
16. Edwards, E. A.: Thrombosis in Arteriosclerosis of the

Upjohn

rheumatoid arthritis

Cortef*



tablets

Available in:

5 mg. tablets in bottles of 50
10 mg. tablets in bottles of 25, 100, 500
20 mg. tablets in bottles of 25, 100, 500

**Registered trademark for the Upjohn brand of hydrocortisone (compound F)*

THE UPJOHN COMPANY, KALAMAZOO, MICHIGAN

Lower Extremities, ed. 1, Springfield, Ill., Charles C Thomas, 1950.
17. Elkins, D. C., and Cooper, F. W.: The effect of vasodilator drugs on the circulation of the extremities, Surgery, 29:323 (Mar.) 1951.
18. Engelberg, H., and Massell, T. B.: Heparin in the treatment of advanced peripheral atherosclerosis, Am. J. M. Sc. 225:1 (Jan.) 1953.
19. Finnerty, F. A., Jr., and Freis, E. D.: Experimental and clinical evaluation in man of hexamethonium (C6), a new ganglionic blocking agent, Circulation 2:828 (Dec.) 1950.
20. Freund, J., et al.: The effect of priscoline on the clearance of radiosodium from muscle and skin of man in normal and diseased limbs, Circulation 8:89 (July) 1953.
21. Friedell, M. T., et al.: Radioactive isotopes in the study of peripheral vascular disease, Arch. Int. Med. 85:667 (Apr.) 1951.
22. Goetz, R. H., and Arnold, P.: Effect of pentamethonium iodide on the peripheral circulation, Lancet 2:408 (Sept. 3) 1949.
23. Gold, H., et al.: Cornell Conferences on Therapy, New York, MacMillan Co., 1953, vol. 6.
24. Green, H. D., et al.: Evaluation of the severity of organic occlusive disease and comparison of the effectiveness of various procedures in relaxing peripheral vasospasm, Circulation 1:1277 (June) 1950.
25. Grimson, K. S., et al.: The effects of priscol on peripheral vascular diseases, hypertension, and circulation in patients, Ann. Surg. 127:968 (May) 1948.
26. Hecbt, H. H., and Anderson, R. B.: The influence of dibenamine on certain functions of the sympathetic nervous system in man, Am. J. Med. 3:3 (July) 1947.
27. Henderson, F. G.; Shipley, R. E., and Chen, K. K.: Pharmacologic studies of 6, 7-dimetboxy-1-(4′-ethoxy-3′-methoxybenzyl)-3-methylisoquinoline, J. Am. Pharm. Ass. (Scientific Ed.) 40:207 (Apr.) 1951.
28. Howard, M. E.: Modern Drug Encyclopedia, ed. 5, New York, Drug Publications, 1952.
29. Katz, R. A.: Impending ischemic gangrene: new nonsurgical therapeutic suggestions, N. Orleans M. & S. J. 98:542 (June) 1946.
30. Keys, A.: Modern health problems in the United States, Minnesota M. 36:7 (July) 1953.
31. Kramer, D. W.: Peripheral Vascular Diseases, ed. 1, Philadelphia, F. A. Davis Co., 1948.
32. Lagen, J. B.: Priscoline as an adjunct for the relief of vasospasm in peripberal vascular disease, Am. Practitioner 1:50 (Jan.) 1950.
33. Lampson, R. S.: Quantitative study of vasoconstriction induced by smoking, J.A.M.A. 104:1963 (June 1) 1935.
34. LeFevre, F.: Management of occlusive arterial diseases of the extremities, J.A.M.A. 147:1401 (Dec. 8) 1951.
35. Littauer, D., and Wright, I. S.: Papaverine hydrochloride, Am. Heart J. 17:325 (Mar.) 1939.
36. McGovern, T.; McDevitt, E., and Wright, I. S.: Theobromine sodium salicylate as a vasodilator, J. Clin. Invest. 15:11 (Jan.) 1936.
37. McIntyre, C., et al.: Tetraethylammonium chloride in evaluation of lower extremity arterial disorders, Surgery 25:348 (Mar.) 1949.
38. Moser, M., et al.: Effect of dibenzyline on skin temperature, peripheral blood flow, and vasomotor responses in normal patients and patients with peripheral arterial diseases, Circulation 8:224 (Aug.) 1953.
39. Murphy, R. A., Jr., et al.: The effect of priscoline, papaverine, and nicotinic acid on blood flow in the lower extremity of man, Surgery 27:655 (May) 1950.
40. Pratt, G.: The present status of sympathectomy in the treatment of vascular diseases, Angiology 1:9 (Feb.) 1950.
41. Rickles, J. A.: Use of intra-arterial aminophylline for peripheral arteriosclerosis, J. Fla. M. Ass. 38:4 (Feb.) 1951.
42. Rogers, M.: Use of a sympathetic drug (priscol), J.A.M.A. 140:272 (May 21) 1949.
43. Rottenstein, H., et al.: The vasodilator effects of priscoline in patients with ischemic extremities, Am. J. M. Sc. 221:669 (June) 1951.
44. Sanders, C.: Cardiovascular and peripheral vascular diseases: treatment by motorized oscillating bed, J.A.M.A. 106:916 (Mar. 14) 1936.
45. Samuels, S. S.: Peripheral Vascular Diseases, ed. 1, New York, Oxford University Press, 1943.
46. Samuels, S. S.: Peripheral arterial disease in the aged, Geriatrics 3:273 (Sept.-Oct.) 1948.
47. Sollman, T.: A Manual of Pharmacology, ed. 7, Philadelphia, W. B. Saunders Co., 1948.
48. Stamler, J.: Pathogenesis and therapy of atherosclerosis, Med. Clin. N. America 36:1 (Jan.) 1952.
49. Texter, E. C., et al.: Evaluation of vasodilator drugs in four patients with arteriosclerosis obliterans, Am. J. M. Sc. 224:4 (Oct.) 1952.
50. Weisman, S., and Allen. E. V.: The failure of histidine and vitamin C and of ether, to improve the peripheral circulation, Circulation 1:127 (Jan.) 1950.
51. Wessler, S.: Medical management of peripheral arterial occlusive diseases, New England J. M. 249:6 (Aug. 6) 1953.
52. Wilkins, R. W., et al.: The effect of the dependent position upon blood flow in the limbs, Circulation 2:373 (Sept.) 1950.
53. Wisham, L. H., et al.: Value of exercise in peripheral arterial disease, J.A.M.A. 153:10 (Sept. 5) 1953.
54. Wright, I. S., and Moffat, D.: The effects of tobacco on the peripheral vascular system, J.A.M.A. 103:318 (Aug. 4) 1934.
55. Wright, I. S.: The treatment of arteriosclerosis obliterans, J.A.M.A. 115:893 (Sept. 14) 1940.
56. Wright, I. S.: Vascular Diseases in Clinical Practice, ed. 1, Chicago, The Yearbook Publishers, Inc., 1948.

ANNOUNCEMENTS

The American Board of Obstetrics and Gynecology announces that the next scheduled examination (Part I) will be held in various cities of the United States on Friday, February 4, 1955. Twenty case abstracts are to be sent to the secretary of the board as soon as possible after receipt of notification of eligibility to the Part I written examination. Candidates are reminded that lists of hospital admissions must accompany new applications and requests for reopening.

Correspondence is to be addressed to Robert L. Faulker, M.D., 2105 Adelbert Road, Cleveland 6, Ohio.

The 24th annual conference of the Oklahoma City Clinical Society will be held at the Biltmore Hotel, Oklahoma City, October 25-28. In addition to scientific sessions the program will include round table luncheons, clinicopathological conferences, dinner meetings, postgraduate panels, and exhibits.

The complete list of speakers may be found in the September issue of the JOURNAL on Page 551.

The third annual Western Cardiac Conference, sponsored by the Colorado Heart Association, Colorado State Department of Public Health, Fitzsimons Army Hospital, Denver VA Hospital, and the University of Colorado School of Medicine will be held in Denver, November 8-13.

Information may be obtained from the Colorado Heart Association, 901 East 17th Avenue, Denver.

The American Urological Association offers an annual award of $1,000 (first prize of $500, second prize $300, and third prize $200) for essays on the result of some clinical or laboratory research in urology. Competition is limited to urologists who have been graduated not more than 10 years and to men in training to become urologists. Details may be secured from William P. Didusch, 1120 North Charles Street, Baltimore, Maryland.

The trustees of America's oldest medical essay competition, the Caleb. Fiske prize of the Rhode

It's Convenient to Buy Kanox

THERAPEUTIC OXYGEN AND ANESTHETIC GASES



Oxygen Therapy
Regulators and Equipment
Repair Service for
Regulators, Flowmeters
and Humidifiers

Nitrous Oxide
Ethylene
Carbon Dioxide
Helium
Oxygen
Cyclopropane

KANSAS OXYGEN, Inc.

Phone 5-5551 HUTCHINSON, KANSAS Box 551

Pure as sunlight



Island Medical Society, announce as the subject of this year's dissertation "Modern Developments in Anesthesia." The essay must be typewritten, double spaced, and should not exceed 10,000 words. A prize of $250 is offered. Information may be secured from the Society, 106 Francis Street, Providence 3, Rhode Island.

The International Academy of Proctology announces the establishment of a teaching and research fellowship in proctology under the direction of Dr. Marcus D. Kogel, dean of the newly formed Albert Einstein College of Medicine, New York City. The academy has voted a $1,000 annual grant for each of three years. Those wishing complete information may write the Academy, 43-55 Kissena Boulevard, Flushing, New York.

Grant From A.M.E.F.

The University of Kansas School of Medicine has been awarded a $29,495 grant from the American Medical Education Foundation, according to a recent announcement. This is the fourth and largest grant the school has received from the fund since 1951, when the first awards were made.

This year's awards, apportioned among the nation's four-year medical schools and six two-year basic science schools, total $2,176,904.71.

The funds received this year will be used to increase salaries of personnel at the Kansas City school, according to Dean W. Clarke Wescoe.

March of Medicine Series

The March of Medicine, television program presented by the American Medical Association and Smith, Kline and French Laboratories, will begin a new series on Sunday, October 31, at 4:30 p.m. The first program will be devoted to the subject of mental illness. The final program in the fall series will be a report of the A.M.A. clinical session in Miami in December. Another series will be presented in the spring of 1955.

A recent survey by the Kansas Academy of General Practice indicates that 99.99 per cent of the Kansas members have complied with an Academy requirement on medical study. The survey covered a three-year period ending December 31, 1953. The Academy also reports that 15 per cent of Kansas members attended the national scientific assembly in Cleveland.

Patronize Journal advertisers.

BOOK REVIEWS

Peripheral Nerve Injuries: Principles of Diagnosis. Second Edition. By Webb Haymaker and Barnes Woodhall. Published by W. B. Saunders and Company, Philadelphia. 333 pages, 272 illustrations. Price $7.00.

The second edition of this monograph continues the high level of excellence previously established. The four sections deal with principles of innervation, examination of the patient, classification of nerve injuries, and, finally, clinical features of the various nerve injuries. The text is lucidly written and clearly illustrated. It is complete without being overly long.

For the surgeon dealing with peripheral nerve injuries, civilian or military, this book is highly recommended.—*C.E.B.*

The Meaning of Social Medicine. By Iago Galdston. Published for The Commonwealth Fund by Harvard University Press, Cambridge. 137 pages. Price $2.75.

This is a rather unusual approach to a current problem couched in a slightly pseudo-sophisticated style and terminology. The author has, according to the example of Don Quixote, set up numerous medical-philosophical windmills with which he has tilted in a comparatively vigorous and effective manner. The term "social medicine," which is never completely defined, is taken to be a description of the complete medicine which considers not only the patient's physiological and mental ailments but also the environmental conditions which have contributed to these or will prevent their cure. It has nothing to do with socialized or state medicine, although the threat is voiced that, unless true social medicine is developed, socialized medicine may result.

The author quite cuttingly observes that this field of medicine is not being adequately presented but avoids explaining why it is not being presented as he would desire. He intimates but never actually admits that the real reason is that there is not enough knowledge in this field. It is his belief that there is need for a revolution in medical instruction in order to accomplish the teaching of so-called social medicine, and he gives little credence to the efforts put forth in present medical school programs. The book could have carried its message in one third of its space and in a simpler form with a more complete perspective and have accomplished a very good purpose since the central theme is based on a sound concept.—*V.E.W.*

Nationally advertised Surgical Supplies and Equipment for your convenience at Topeka, Joplin, Kansas City, St. Joseph

GOETZE NIEMER CO

Traditions established during 60 years management by Dr. W. F. Goetze (AMA) assures intelligent servicing of your orders

PATRONIZE JOURNAL ADVERTISERS

It is advertisers who make possible the publication of the Journal in its present form.

They are deserving of your patronage.

UNIVERSITY OF KANSAS SCHOOL OF MEDICINE

Postgraduate Medical Course

INTERNAL MEDICINE

November 15, 16, 17 & 18, 1954

Guest Instructors:

WENDELL H. HALL, M.D., Ph.D., Section of Special Infectious Diseases, Minneapolis Veterans Administration Hospital; Clinical Assistant Professor of Medicine, University of Minnesota School of Medicine, Minneapolis.

EDGAR A. HINES, JR., M.D., Consultant in Medicine, The Mayo Clinic; Associate Professor of Medicine, University of Minnesota Graduate School of Medicine, Rochester.

EBBE C. HOFF, M.D., Professor of Neurological Science and Lecturer in Physiology, Medical College of Virginia, Richmond.

CHESTER S. KEEFER, M.D., Special Assistant to the Secretary of the U. S. Department of Health, Education & Welfare, Washington, D. C.; Wade Professor of Medicine, Boston University School of Medicine, Boston, Mass.

JOSEPH B. KIRSNER, M.D., Ph.D., Professor of Medicine, University of Chicago School of Medicine, Chicago, Ill.

DAVID W. TALMAGE, M.D., Assistant Professor of Medicine, University of Chicago School of Medicine, Chicago, Ill.

THOMAS G. WARD, M.D., Associate Professor of Microbiology, Johns Hopkins University School of Hygiene & Public Health, Baltimore, Md.

WALTER L. WINKENWERDER, M.D., Assistant Professor of Medicine, Johns Hopkins University School of Medicine, Baltimore, Md.

ERIC E. WOLLAEGER, M.D., Consultant in Medicine, The Mayo Clinic; Associate Professor of Medicine, University of Minnesota Graduate School of Medicine, Rochester.

LEWIS W. ANDREWS, Executive Director, Kansas State Commission on Alcoholism, Topeka.

Subjects to Be Discussed:

Infectious Diseases

Present Status of Poliomyelitis Vaccines and Gamma Globulin.
Bacterial Pneumonias.
Influenza.
Common Cold.
Histoplasmosis, Blastomycosis, Actinomycosis.
Tuberculosis.
Brucellosis, Acute and Chronic.

Peripheral Vascular Diseases

Physical Examination of Patient.
Special Diagnostic Studies.
Treatment of Chronic Venous Insufficiency.
Medical and Surgical Treatment of Chronic Occlusive Arterial Disease of Lower Extremities and of Popliteal Aneurysms.

Allergies

Action of Antibiotics on Host and Parasite.
Fundamental Concepts of Immunity in Allergy.
Role of Immunization in Preventive Medicine.
Comments on Diagnosis of Allergic Diseases, Including Present Status of Skin Testing.
The Mechanism of Action of ACTH and Cortisone in Allergic Diseases.
Management of Allergic Diseases Including Use of ACTH-Cortisone

Gastroenterology

Antibiotics in Gastroenterology.
Steatorrhea and Sprue.
Anticholinergic Drugs in the Management of Gastrointestinal Disease.
Medical Management of Regional Enteritis.
Proctoscopy (Motion Picture).
Psychiatric Approaches to Treatment in Ulcerative Colitis.
Medical Management of Ulcerative Colitis.
Chronic and Relapsing Pancreatitis.

Alcoholism

Management of the Chronic Alcoholic.
Activities of the Kansas State Commission on Alcoholism.

Dinner Meeting—Tues., Nov. 17: The Future of the Private Practice of Medicine, Dr. Chester S. Keefer.

Demonstrations:

Physical Examination of Patient for Peripheral Vascular Disease.
Posterial Tibial Block, Lumbar Block, Stellate Block.
Liver Biopsy, Esophageal Dilatation, Bone Marrow Biopsy.

The guest instructors will participate in Panel Discussions and Ward Rounds, which are scheduled with each section of the program.

Coronary Heart Disease in Young Adults. A Multidisciplinary Study. By Menard M. Gertler, Paul D. White, and others. Harvard University Press, Cambridge 38, Massachusetts. 218 pages, 43 illustrations, 61 tables. Price $5.00.

This book presents a study of 100 patients with myocardial infarctions before the age of 40. Statistical comparisons are made with a matched control group of normal patients. They are duplicated as accurately as possible in pairs regarding age, sex, height, weight, body build, race, and occupation. Coronary heart disease is evaluated clinically from the following viewpoints: sex selection, and the morphological, genetic, athletic, occupational, physiological, psychological, clinical, dietary, hormonal, and biochemical aspects.

One clinical impression gained is that as a group patients with coronary disease appear about 10 years older than their chronological age. Although coronary patients consumed some forms of alcohol and used tobacco in statistically larger amounts than the control group, findings did not warrant any conclusions as to a causal relationship between the use of alcohol or tobacco and coronary heart disease. The morphological characteristic of the young coronary heart disease patient is most often that of an endomorphic mesomorph. The mean levels of serum total cholesterol in the coronary heart disease group are higher than the matched control group. The distribution curves are markedly continuous, however.

It is inferred that a threshold level of serum total cholesterol for coronary heart disease does not exist and that additional factors probably play a role. One of these appears to be the serum phospholipids. The ratio of total cholesterol to lipid phosphorus is significantly higher in the coronary group. The ratio is thought to be related to the colloidal stability of cholesterol in serum, which in turn is related to the amount of cholesterol deposited in the intima of coronary vessels. It is concluded that the coronary heart disease group ingests less cholesterol than the control group but has more cholesterol in the serum. It appears that an endogenous source of cholesterol plays a definite role in the etiology of coronary heart disease.

It is hoped through such studies that the ability to prevent coronary disease will be evolved by determination of etiology and pathogenesis of the disease process.—*F. N. L.*

Practical Fluid Therapy in Pediatrics. By Fontaine S. Hill. Published by W. B. Saunders Company, Philadelphia. 275 pages, 9 photographs. Price $6.00.

Dr. Fontaine has produced a book which has long been needed by the physician dealing with infants and children. The book is divided into three sections: basic physiological principles and their clinical significance, diagnosis and treatment of clinical conditions, and the technical procedures. The author has written in such a way that a complete review of pathophysiology in fluid and electrolyte balance may be understood by the average general practitioner and pediatrician.

The summary and clinical applications at the end of each chapter aid one in comprehending the reasons for proper fluid therapy. Diagrams, graphs, and charts, as well as case presentations, are used freely to emphasize points which otherwise may not have been clear. The applicability of this book for a reference for the medical student, pediatric resident, and other physicians treating and caring for children is excellent; it should be a superb addition to one's library.—*A. M. D.*

A.M.A. Fundamentals of Anesthesia. Third Edition. Published by W. B. Saunders Company, Philadelphia. 279 pages, 89 illustrations, 19 tables. Price $6.00.

This book was prepared under the editorial direction of the Consultant Committee for Revision of Fundamentals of Anesthesia, Council of Pharmacy and Chemistry of the A.M.A.

All of the good of the first two editions of this important publication has been retained with much important information added, thus making it an outstanding text for the beginning or occasional anesthetist. Although each chapter has been enlarged into a book by various and sundry authors, this presents the best of each in a compact form.

Obviously the dogma which seems to be present is necessary as the details have been deleted. Fortunately none but the newest of drugs, which aren't in everyday use, were omitted and the comment on the pharmacology and technic of using all drugs is adequate. Some prejudice is obvious, such as the condemnation of chloroform because of its potency, with no statement as to potency of trichloroethylene.

This will undoubtedly satisfy the urgent need for a text for students of anesthesia. Because the intent of the authors was to satisfy this need, criticism for inadequacies due to lack of detail is unjustified.—*E. L. F.*

Electrocardiography. By E. Grey Dimond. Published by C. V. Mosby Company, St. Louis. 61 pages, 272 illustrations. Price $14.00.

This volume might well be called a self-teacher of electrocardiography. It begins with such funda-

Medical and Surgical Supplies for Doctors of Medicine and Hospitals

Munns Medical Supply Co.
512 Kansas Avenue
Topeka, Kansas

A Mutual, Open-end, Diversified Investment Fund with Full Management Discretion

MUTUAL TRUST

Offered by
MUTUAL DISTRIBUTORS, Inc.
1016 Baltimore • Kansas City 5, Mo.
Principal Underwriter and Distributor

PROSPECTUS ON REQUEST

PLEASE SEND ME PROSPECTUS

*Name*_____

*Address*_____

*City*_____ *State*_____

Clarence W. Glassen, State Representative



Woodcroft Hospital--Pueblo, Colorado

A private hospital for the scientific treatment of neuro-psychiatric disorders, including alcoholism and drug addiction. Beautiful landscaping and home-like surroundings afford a restful atmosphere. Accommodations vary from single rooms with or without bath to rooms en suite, allowing for segregation of guests.

Detailed information furnished on request.

KARL J. WAGGENER, M.D.

mental considerations as the types of machines used in recording the electrocardiograph and the advantages and disadvantages of various types. Comments regarding eradication of interfering influences and technical errors in the electrocardiogram are described in detail with good illustrations. The reader is then guided carefully in a stepwise fashion by graphic illustrations, photographs, and diagrams, together with a lucid text from the fundamentals of electrocardiography, into a more sophisticated realm of controversial matters in electrocardiographic interpretation.

We believe that the novice can, with the aid of this volume, teach himself the fundamentals of electrocardiography and acquire an ability to interpret correctly all but the most unusual electrocardiographic abnormalities. Although the book is valuable as a reference, it should be read in its entirety, and such study could best be done by self-conducted exercises of blackboard drill and interpretation of electrocardiographic tracings with clinical knowledge of the patient.—*M. S. A.*

Refresher Course on Injuries

Practically all general practitioners, urban or rural, handle "on-the-job" injuries and illnesses daily, even though they may not consider that they are engaged in industrial practice. Ordinarily they probably would not be interested in a scientific program devoted to industrial medicine. But they should find considerable interest in a refresher course devoted to persons who come into their offices with injuries such as contact dermatitis from spray chemicals used around the home and farm and illnesses that may result from occupational hazards. If the course also included better methods of handling reports and collections on cases covered by insurance, interest would be even greater.

The University of Kansas School of Medicine has planned just such a course for December 6, 7 and 8. It is designed to meet the problems of the general physician who must be prepared to handle all types of accidents and sickness affecting people while engaged in their work. The continual increase in the number and complexity of mechanical equipment and chemicals used around the home, on the farm, and in many small businesses, increases the demand on the family doctor for what is ordinarily considered industrial medicine. The entire program of this course is built around that phase of general practice, with one day devoted to occupational hazards. The second day takes up such medical aspects as poisonings, dermatitis and dust, and the last day covers the field of trauma and surgery.

A copy of the complete program is available upon request from the Department of Postgraduate Education, University of Kansas Medical Center, Kansas City 12, Kansas.

Rural Health Program

The A.M.A. Council on Rural Health and its advisory committee, made up of representatives from several national, agricultural and educational organizations, held a three-day meeting in Chicago recently and formulated a three-point program on which the council will concentrate during the next year. The three points are:

1. More doctor participation with lay groups. Rural people are asking for and welcoming assistance and advice.
2. More cooperation with country newspapers and farm journals, in supplying health information.
3. Bringing rural people closer together by suggesting that county medical societies invite county extension agents and farm leaders to appear on their programs and that rural organizations invite physicians to speak to them.

Plans for the forthcoming Tenth National Conference on Rural Health were formulated. This annual conference is to be held at the Schroeder Hotel, Milwaukee, February 24-26, 1955. As in previous years, the Thursday morning session preceding the formal opening of the conference will be for physicians. This meeting will be devoted to problems confronting physicians who are members of state rural health committees or committees handling rural health programs. The formal sessions of this conference will begin Thursday afternoon and continue through the Saturday luncheon session.

Twins in Heredity Study

Thirty-five pairs of twins from 19 states and Canada responded to a recent appeal for information on identical twins afflicted with multiple sclerosis, according to the National Multiple Sclerosis Society. Preliminary studies have been started on the possible hereditary and environmental causes of the disease.

Initial work is to establish that the twins are identical and that one or both has multiple sclerosis. For purposes of research, the "ideal" twins would be those sets where one member has the disease and the other has not. Both would be studied to try to determine factors which may have been responsible for the disease in the afflicted twin.

Twins who wish to volunteer information as subjects for research are asked to write the National Multiple Sclerosis Society, 270 Park Avenue, New York 17, New York.

THE LATTIMORE-FINK LABORATORIES

Topeka — El Dorado
Kansas

J. L. Lattimore, A.B., M.D., Pathologist
A. A. Fink, A.B., M.D., Pathologist
H. C. Ebendorf, M.T., Serologist
A. C. Keith, B.S., Chemist
L. W. Hull, A.B., Bacteriologist
Walter Norris, A.B., Chemist

Anatomical and Clinical Pathology

Containers Furnished Upon Request

Would You Feel Safer With

HIGHER LIMITS

of

PROFESSIONAL LIABILITY PROTECTION— WITHOUT ANY COVERAGE RESTRICTIONS?

What is adequate for you—
$25,000, $50,000, $100,000?

More than 700 of your colleagues in Western Missouri and Kansas have already chosen the worry-free dependability of the famous U. S. F. & G. Comprehensive Liability Policy for Professional Men.

Call George Staebler, Jr.

The Meade Company Insurance

119 West 6 Topeka 4-0537

Deformity Appliances of Quality

Orthopedic and Surgical Appliances

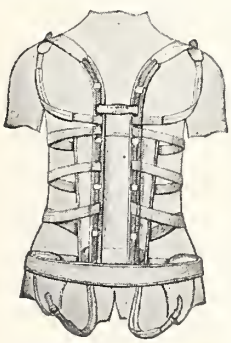
Artificial Limbs
Trusses

Abdominal Supports

Elastic Hosiery

Foot Supports

Surgical Corsets



Taylor Back Brace
Made to Order in
Our Own Factory

P. W. HANICKE MFG. CO.
1009 McGee St. Victor 4750
KANSAS CITY, MO.

THE
MEDICAL PROTECTIVE
COMPANY
FORT WAYNE, INDIANA

PROFESSIONAL PROTECTION
EXCLUSIVELY
SINCE 1899

specialized service
assures *"know-how"*

TOPEKA Office:
J. E. McCurdy, Rep.,
1035 Randolph Avenue,
Telephone 2-3027

Polio Research Grant

The University of Kansas School of Medicine recently received a research grant of $105,111 from the National Foundation for Infantile Paralysis. The fund makes possible continuation of a search for modified strains of polio virus which could be useful in providing an improved vaccine against the disease. Dr. Herbert A. Wenner, pediatrics research professor, will direct the study.

Bulletin on Rheumatic Diseases

The first four volumes of the *Bulletin on Rheumatic Diseases* have been bound in soft cover and are available to physicians at a cost of $1.00. Requests are to be sent to the Arthritis and Rheumatism Foundation, 23 West 45th Street, New York 36, New York.

The publication, issued nine times a year, is available without charge to physicians and medical students who ask that their names be put on the mailing list.

Information on Medical Scholarships

Requests for information on available medical scholarships and loans for a medical education have prompted the publication of a comprehensive new pamphlet on the subject by the A.M.A.'s Council on Rural Health in co-operation with the Department of Public Relations. Scheduled for release in April, this booklet will contain a compilation, by states, of pertinent information regarding all types of medical scholarships and loan funds now available through medical society, governmental, and other special funds. Copies may be obtained from the Council on Rural Health.

Although approved medical schools are now accepting their largest freshman classes—almost 7,500 students—the number of applicants for admission to medical schools has decreased for the fourth consecutive year. The freshman class of 1953-1954 had some 2,085 fewer applicants than the previous year's class, and almost 10,000 fewer individuals are making application now than did in 1949-1950 when the GI bill was in full force.

Historical Material Needed

In preparation for the observance of The Kansas Medical Society's centennial anniversary, members of the Committee on History are attempting to collect all material of historical interest. Physicians who can contribute information, records, etc., are urged to send such to

Committee on History
Kansas Medical Society
315 West 4th Street
Topeka, Kansas

CLASSIFIED ADVERTISEMENTS

FOR SALE—Modern office equipment of deceased physician. Used only four years. Equipment for general practice, including x-ray. Write the Journal 15-54.

FOR SALE—Castle autoclave size 8 inches by 16 inches, practically new, priced right. Write the Journal 19-54.

WILL GIVE most all office equipment and furnishing to a younger doctor who will locate in my town of 3,500 population for general practice. Retiring because of age. Write the Journal 20-54.

LABORATORY TECHNICIAN, registered, wanted for midwestern medical group of eight. Excellent facilities and liberal salary. Write the Journal 21-54.

FOR SALE—EENT practice and office equipment. Established 34 years in college town. Leaving because of wife's health—Write the Journal, 16-54.

FOR SALE—General Practice in small north central Kansas community, office and office equipment available. Residence for rent. Write the Journal, 17-54.

FOR SALE, Due to business change:
1 Brown-Burger Operating and Catheterizing Cystoscope—Excellent.
1 Infant Brown-Burger Single Catheterizing Cystoscope—New Condition.
1 Ophthalmoscope and Can—New.
1 Large Burglar Proof Bank Safe—Double Time Lock and Day Combination.
200 K.V. Westinghouse X-ray Machine, Rotary Anode Tube, Spot Film—Excellent.
Reply to the Journal, 18-54.

The Neurological Hospital

2625 West Paseo,

KANSAS CITY, MISSOURI

★ ★ ★

A voluntary hospital providing the care and treatment of nervous and mental patients and associate conditions.





PARKE-DAVIS speaks to the public...

IAT people think about doctors is tty important to the future of the ctice of medicine in this country.

Can the power and influence of ertising—the *right kind* of adver- ng—be employed to bring home people what the physician of today really do for them, if they'll only him the opportunity?

Parke, Davis & Company's answer this question is their "See Your ctor" advertising program which y started twenty-six years ago and e been carrying on ever since. h message in this continuing series phasizes the same major theme: *importance of prompt and proper lical care.*

No products are mentioned; that he province and responsibility of physician.

Because these messages are all "picture stories" that dramatize the informative and serious material they present, they are among the best-read advertisements being published today. Above everything else, we try for plausible, believable messages that will nudge the reader into action without either raising false hopes or scaring him. We want him to have not only increased confidence in his doctor, but in the professional background and skill of the pharmacist who fills the prescription, and in the medicine itself.

We naturally hope that the reader will come to know and recognize Parke-Davis as a leader in a fundamental American industry, and to associate our name and label with manufacturing skill, careful testing, and enlightened research.

A program of this kind, if it is to do the greatest good, must be brought to the attention of millions of people. That is why the "See Your Doctor" messages have appeared and are currently published in the SATURDAY EVENING POST, LIFE, TIME, NEWSWEEK, TODAY'S HEALTH, and other leading magazines.

While the broad problem is one which admittedly challenges the skill and resourcefulness of many organizations that have the interest of Medicine at heart, Parke-Davis is proud to have a part in pioneering and developing a type of advertising approach which is proving increasingly effective in meeting this challenge. *PARKE, DAVIS & COMPANY, DETROIT 32, MICHIGAN.*

MEDICAMENTA VERA

TABLE OF CONTENTS

NOVEMBER, 1954

ORIGINAL ARTICLES

Medical Care in Pediatric Surgery—Frederic Gerard Burke, M.D., Washington, D.C. . 621

A General Practitioner Studies Psychiatry—Prescott W. Thompson, M.D., Topeka, Kansas 624

Congenital Absence of the Vagina: Review and Case Report—Stanley L. VanderVelde, M.D., and Joseph J. Hovorka, M.D., Emporia, Kansas 628

The Widening Scope of the State Hospital, with Special Reference to Geriatric Medicine and Surgery—C. Maxwell-Joyner, M.D., Larned, Kansas, and Rudolf Hofbauer, Wichita, Kansas 631

EDITORIALS

Is It Art? 635

Federal Aid in Kansas 635

Annual Meeting in Hutchinson 636

MISCELLANEOUS

President's Page 634

Clinicopathological Conference 638

Some Basic Concepts of the Acute Radiation Syndrome—Senior Thesis—Eugene T. van der Smissen, M.D. 656

Entered as second-class matter at the post office at Fulton, Missouri, June 19, 1953, under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of October 3, 1917, authorized on July 2, 1918. Office of Publication: 1201-05 Bluff Street, Fulton, Missouri.

THE JOURNAL OF THE KANSAS MEDICAL SOCIETY

Editorial Board

ORVILLE R. CLARK, M.D., *Editor,* Topeka

JOHN W. CAVANAUGH, M.D., Topeka

DAVID E. GRAY, M.D., Topeka

RICHARD GREER, M.D., Topeka

DWIGHT LAWSON, M.D., Topeka

Associate Editors

DONALD P. TREES, M.D., Wichita

GLEN R. SHEPHERD, M.D., Kansas City

Information

Ownership: The Journal is a non-profit publication owned and published monthly by the Kansas Medical Society.

Subscription: A year's subscription to the Journal is included in membership in the Kansas Medical Society, with $2.00 of each member's dues apportioned to the Journal. Rates to others, except in foreign countries, $4.00 per year or 60c per copy.

Material: Scientific articles, editorials, and data of general interest are invited from all members. Articles are to be submitted on condition that they are contributed solely to this publication. A right is reserved to reject any material deemed unsatisfactory.

Manuscripts: Only manuscripts that are typewritten on one side, double spaced, and original copies can be accepted. Manuscripts will be returned upon request.

Advertising: All advertising contracts, and all copy from advertisers under contract are subject to approval of the editorial board. Copy should be received by the 20th of the month immediately preceding the month of publication.

Non-Responsibility: Although an effort is made to publish only accurate articles and legitimate advertisements, the Journal denies legal responsibility for any statements, opinions, or advertisements appearing under the names of contributors or concerns.

PAULINE FARRELL, Managing Editor and Advertising Manager

OLIVER E. EBEL, Business Manager

THE KANSAS MEDICAL SOCIETY

President . . . . . MURRAY C. EDDY, M.D., Hays

Past President . . . . . LUCIEN R. PYLE, M.D., Topeka

President-Elect . . . . . JOHN M. PORTER, M.D., Concordia

First Vice-President . . . CLYDE W. MILLER, M.D., Wichita

Second Vice-President, CONRAD M. BARNES, M.D., Seneca

Constitutional Secretary, JAMES A. BUTIN, M.D., Chanute

Treasurer . . . . . JOHN L. LATTIMORE, M.D., Topeka

A.M.A. Delegate, 1954-1955, GEORGE F. GSELL, M.D., Wichita

A.M.A. Delegate, 1955-1956, LAURENCE S. NELSON, M.D., Salina

Councilors and Councilor Districts

First District, FREDERICK E. WRIGHTMAN, M.D., Sabetha

Second District . . . GLENN R. PETERS, M.D., Kansas City

Third District . . . . . H. PENFIELD JONES, M.D., Lawrence

Fourth District . . . . CHARLES E. VESTLE, M.D., Humboldt

Fifth District, SEVERT A. ANDERSON, M.D., Clay Center

Sixth District . . . . . FLOYD C. TAGGART, M.D., Topeka

Seventh District . . . . . TO BE ANNOUNCED

Eighth District . . . . . JAMES E. HILL, M.D., Arkansas City

Ninth District . . . . LAURENCE S. NELSON, JR., M.D., Salina

Tenth District . . . . . HAROLD M. GLOVER, M.D., Newton

Eleventh District . . . . NORTON L. FRANCIS, M.D., Wichita

Twelfth District . . . . . CYRIL V. BLACK, M.D., Pratt

Thirteenth District . . LLOYD W. REYNOLDS, M.D., Hays

Fourteenth District . . . . JUSTIN A. BLOUNT, M.D., Larned

Fifteenth District . . . . ROBERT G. KLEIN, M.D., Dodge City

Sixteenth District . . . . . JAMES L. JENSON, M.D., Colby

Seventeenth District . . . . . H. PRESTON PALMER, M.D., Scott City

OLIVER E. EBEL, EXECUTIVE SECRETARY

RUEBEN M. DALBEC, EXECUTIVE ASSISTANT

Executive Office: 315 West Fourth Street, Topeka

THE JOURNAL
of the
KANSAS MEDICAL SOCIETY

Copyright, 1954, by The Kansas Medical Society

Volume LV NOVEMBER, 1954 No. 11

Medical Care in Pediatric Surgery

Frederic Gerard Burke, M.D.
Washington, D. C.

Infants and children are frequently regarded as miniature men and women, both in the general practice of medicine and in surgery. Consideration of the factors of immaturity and growth and development attest to the incorrectness of this viewpoint. During the first few years of life, the patient is characterized by markedly immature but very active physiologic processes and rapid metabolism, necessitated by the acceleration of growth. There is a particular instability of the mechanisms that control water, acid-base, and nitrogen balance.

The younger and more immature the infant or child, the more rapid the extensive changes that occur in physiochemical disturbances of the normal balance of tissue fluids. Normally, the quantity of circulating plasma is remarkably constant, but under conditions of disease, changes in the volume and composition closely reflect the changes of the fluids in other compartments. There are many mechanisms with a remarkably standard pattern under abnormal states which are activated to conserve body fluids and minimize loss of water. These changes are characterized primarily by shifts in the concentration of the various water holding salts within their respective compartments.

Since serious surgical disorders result in dehydration, alkalosis, acidosis, and in anemia, the pediatric surgeon and the pediatrician must reckon with these changes in the management of a sick child if optimal results are to be obtained. As important as these factors are in the preparation of the adult for surgical procedures, the liability and immaturity of these balancing mechanisms in children make their adjustment of greater importance. Of course the arithmetic majority of surgical procedures performed in children, such as hernia repairs and eye, ear, nose and throat operations, seldom require any parenteral fluid therapy. Indeed, minor shifts of acid-base and fluid balance are best "intelligently neglected" since the body reserves can be depended upon to compensate satisfactorily any minor shifts. When the oral intake is reduced or fluid losses are excessive, as by vomiting, enterostomy, diarrhea, fever, etc., then these losses must be replaced, usually by parenteral routes. In general, there is more hazard in over hydration by excessive and enthusiastic infusions of fluids in surgical disorders in children than in not supplying enough; so great care and close observation must be employed in supplying parenteral repair solutions.

Physiologic salt solution is frequently used in fluid replacement therapy of children, and if the kidneys are functioning normally, it helps to correct alkalosis or acidosis. Excessive amounts, particularly in young babies and in other children with decreased renal function, may lead to edema on the basis of sodium retention.

The various modifications of Ringer's solution with magnesium, calcium, and potassium added are most valuable in correcting water and salt loss. Hartmann's solution contains sodium lactate in addition to the other ions of Ringer's solution and is more effective in correcting acid-base imbalance associated with dehydration. Glucose should be used in conjunction with Hartmann's-Ringer's solution when ketosis is present, and it is well tolerated and utilized as a 10

Presented at the 75th annual session of The Kansas Medical Society, Topeka, May 2 to 6, 1954, in panel discussion on Pre- and Postoperative Care of Infants and Children. Participating in this panel were Frank L. Menehan, M.D., Wichita, Moderator, and David R. Akers, M.D., Denver.

Professor of Pediatrics, Georgetown University, Washington, D. C.

per cent solution when given intravenously or as a 5 per cent solution subcutaneously. Five or 10 per cent glucose with Hartmann's solution added is probably the safest parenteral fluid employed in helping restore the mineral and caloric imbalance in pre and postoperative care, when the need for fluid and minerals has been determined.

Whenever possible, the oral route for hydration is by far to be preferred, particularly in reference to caloric restoration. Fluids can also be supplied by rectum such as retention enemas and subcutaneously with the assistance of hyaluronidase to facilitate absorption. Whenever parenteral fluids must be employed over a period of several days, there is considerable advantage to a cut down with the insertion of polyethylene tubing, which permits much greater and more efficient control over the fluid intake.

The effects of hypoproteinemia are many and well known. The hypoproteinemic child is more susceptible to infection, and when infection supervenes the course is apt to be stormy and protracted. Children who, prior to development of a surgical disorder, are suffering from a protein deficiency, develop an aggravation of this condition during the disease process and may succumb even in the face of bacteriologic control by antibiotics because of the severe decrease of tissue and plasma proteins. Furthermore, such a child is more susceptible to shock, and postoperatively he may suffer from delayed rate of healing. Also, his chance of developing a postoperative adynamic ileus is increased. Stomal difficulties, chronic anemia, and intestinal obstruction are more likely to occur when blood proteins are low. The development of ascites and the effects of hypoproteinemia in hemorrhage, burns, peritonitis, and obstruction are well known.

When possible, protein should be supplied by mouth. In infants, when the intake falls below 2.2 grams per kilogram of body weight, a negative balance is to be expected.

Amino acid solutions may be used to control and maintain nitrogen equilibrium, usually as a 3½ per cent solution combined with dextrose, and may be given to children who for one reason or another cannot take adequate amounts of protein by mouth. Of greater value than amino acid solutions is human plasma or human albumin to help restore normal protein values of the blood. They are particularly useful when the alimentary tract is obstructed as in atresias, pyloric stenosis, intussusception, volvulus, peritonitis, or when the intestinal tract should be kept at rest during the postoperative course of abdominal surgery. The value of plasma and human albumin in restoring circulating volume while raising the intravascular osmotic pressure is well known in the management of the adult surgical patient and is equally valuable in children.

Blood transfusions are indicated for the replacement of blood lost by hemorrhage and are considered essential when the hemoglobin content is below 9 grams per 100 cc. Blood loss at the time of operation, if it is more than a minor degree, should be replaced before the patient leaves the operating room. The frequently noted beneficial effects of small repeated blood transfusions in the postoperative course of children convalescing from a serious surgical disorder justify the use of blood even though the hemoglobin and red blood cell counts have slightly higher values. This effect has been attributed in part to naturally occurring antibacterial factors in adult blood in addition to its immediate value as a valuable source of protein, minerals, and oxygen-carrying properties of the red blood cells.

Vitamin K should be administered to the newborn before and after surgical procedures to help circumvent hemorrhagic diathesis, and attention should be paid to the calcium and Vitamin C intake, particularly in early infancy.

Because of the ever present danger of concurrent or postoperative infection, the use of antibiotics and chemotherapeutic agents just before and following major surgical procedures is frequently advisable. When the hazard is respiratory infections, a single injection of a long acting, depo-penicillin such as bicillin may have considerable prophylactic benefit. Terramycin or chloromycetin may provide considerable protection when bowel surgery is performed. Whenever time permits in the instance of elective intestinal surgery, such as in Hirschsprung's disease, reduction in the bowel flora is recommended by the use of the broad spectrum antibiotics.

Morphine as preoperative preparation before anesthesia has been much maligned in pediatric practice, and the dangers have perhaps been unduly emphasized. In small doses effective relaxation is possible with a safety margin that is not too narrow. It is not necessary in infants up to the age of 4 or 5 months, but after 6 months it is a valuable aid in securing relaxation. The doses recommended begin at 1/144 of a grain at the 6-month level and increase to 1/48 of a grain at the 3 to 5-year level. Where there might be difficulty in securing these small graduated doses, the danger of central respiratory inhibition, due to the possibility of using a greater dosage than is necessary, might constitute a contraindication to its use.

The preoperative use of atropine or scopolamine in combination with morphine is of considerable assistance in providing a smooth anesthetic. Fear and apprehension are diminished, secretions are reduced in amount, untoward and dangerous vagal reflexes are rendered less hazardous, and the induction is made smoother. Whenever possible, barbiturates should be given the night before the operation and 1 to 2 hours

preoperatively to help reduce psychic trauma associated with the operative procedure.

Conservation of body heat in young infants during the operative procedure is a point which cannot be overemphasized. The use of an electric heating pad or hot water bottles around the infant's body, particularly during prolonged operations, reduces the chances of shock and minimizes the danger of postoperative infection. Oxygen therapy and measures to maintain a normal body temperature are important supportive procedures, as are suction facilities for the nose, throat, and stomach during the period of time immediately after the operation.

These points that have been mentioned are general points which are of particular interest to the pediatrician in his phase of responsibility in the management of a surgically sick child. In each or any specific condition, any phase of surgical stress may assume particular or peculiar significance. The best preoperative preparation in the world is a poor substitute for early and accurate diagnosis. For example, in pyloric stenosis, the relative safety and success of surgical correction is attested to by its general acceptance as the method of choice for the treatment of this condition. In adequate hands, the mortality rate attendant to this operaion should be no higher than 1 per cent. Consideration of the physiochemical equilibrium and fluid balance of these infants before and after operation would give promise that this mortality will be kept at a low rate.

In no instance should an infant be operated upon for pyloric stenosis without satisfactory preoperative preparation. This consists of adequate hydration, correction of any existing anemia by blood transfusion, and correction of alkalosis by the parenteral administration of Ringer's solution and glucose. Probably the two most important factors responsible even for the existing mortality are failure to adequately prepare these patients for operation and failure to institute surgical procedures early.

It is well known that severe alkalosis can result in generalized convulsions and in fatal depression of respiration in the small infant. The tetany that results from alkalosis should be relieved by restoration of acid-base balance and the intravenous administration of calcium gluconate. This balance can nearly always be restored by the intravenous administration of salt solution. Surgical correction of pyloric stenosis is never an acute emergency, and restoration of homeostasis and caloric equilibrium is essential to its successful outcome. Because these infants are actually in a state of starvation, external heat should be constantly applied before, during, and after operation. Formulas should be withheld for at least 12 hours because of the frequency of postoperative vomiting, and at that time half-strength breast milk or a weak acidified evaporated milk formula can be employed in small amounts at three- to four-hour intervals.

One final area of interest to the pediatrician which is of considerable importance in elective pediatric surgery is selection of the time to operate. While the location and nature of the defect that clearly indicates prompt surgery offers no problem in timing, there exists a difference of opinion as to when to operate when conditions exist that apparently do not offer an immediate threat to life.

An example is the inguinal hernia. Most textbooks still have elaborate diagrams of ingenious devices calculated to truss the anatomically weak point in the inguinal region. Trusses for this purpose probably have no valid use and should be discontinued except in the rare case. There is a bad psychologically deterrent effect upon the young mother who lives in constant fear of strangulation of the hernia, which has its effect not only on the parents but also on the infant. Too often these babies are pampered and given everything they want or think that they want to prevent them from crying, the parents appreciating that the increased intraabdominal pressure resulting from crying causes the hernia to descend. While an occasional inguinal hernia will spontaneously heal during the first year, it is doubtful that any inguinal hernia will heal spontaneously after the first year. Even if incarceration has not occurred before the 12th month, surgical repair should certainly be instituted. If incarceration occurs any time after birth, surgical repair, not a truss, is the correct management. In most cases today, inguinal hernias should be surgically repaired when it is definitely ascertained that one is present.

Plastic surgery, cardiothoracic, urologic, and orthopedic defects all offer specific problems in timing, and consideration of the peculiar factors of growth and development, not only of the body but also of the personality, that characterize the infant and child should help in the selection of the time for surgical correction.

In summary of this general outline of some of the principles of medical management of the infant or child who is a candidate for a surgical operation, it is well to keep in mind that the operation represents only one specific phase in a treatment regime that does not lose sight of the patient as a whole. As important as good surgical technique is to the successful outcome, failure to give keen attention to medical factors offers a serious threat to good results.

These factors are:

1. Hydration. This consists of the administration of parenteral fluids, if the oral route is not available, with particular reference to selection of the correct fluid, route, rate, and technique of administration.

2. Restoration and maintenance of caloric, nitrogen, and acid-base balance.

3. Measures to conserve body heat, relieve apprehension and pain, and prevent psychic trauma by the use of barbiturates and morphine.

4. The restoration of a normal blood picture by the use of small, repeated blood transfusions.

5. Measures calculated to control and prevent infection by the use of antibiotics.

6. Symptomatic and supportive therapy as indicated by the particular case, including the use of oxygen and suction facilities for the nose, throat, and stomach.

7. Consideration of the factors of growth and development to assist in the selection of the best time for the institution of elective surgery.

A General Practitioner Studies Psychiatry

Prescott W. Thompson, M.D.
Topeka, Kansas

INTRODUCTION

During the Kaw River flood in 1951 the author, who at that time had completed two years of psychiatric training after eight years of practice in general medicine and surgery, was again temporarily thrust into a busy general practice in a Topeka dispensary caring for flood victims. In reflecting upon this experience, this former general practitioner became aware of how much psychiatry can add to the physician's understanding of the ailments of many patients.

This paper will present five case examples, drawn from the brief dispensary experience, in which the physician's psychological awareness, nurtured by additional training, provided the clue to diagnosis or to further understanding of the patient's problem. The emergency nature of the dispensary was such that follow-up was not possible, and this paper therefore can only concern itself with what the author considered to be a broadening of his own conceptual sphere and various observations and suggestions drawn from this consideration.

CASE REPORTS

Case 1. A 64-year-old woman came to the dispensary to request a medical order that she be served her meals on a tray. She stated that weakness in her legs made it difficult for her to stand in line in the dining room. The nurse (to whom she had first complained, as was customary in the dispensary) was impatient with the woman's request for special privilege, particularly since the patient did not appear acutely ill and had indeed walked to the dispensary without help.

The doctor observed a weather-beaten old woman who sat quietly and spoke only to answer direct questions, except to volunteer that she thought one tray a day would be enough for her. Her facial expression was paradoxical, with a troubled forehead but a steady smile.

It was noted that her pulse was slow, whereupon she stated that she had been taking digitalis for several years. She had, however, before the flood, managed her home, done all her own shopping, and chopped wood. Except for the leg weakness, she had experienced no recent symptoms of cardiac decompensation. A general physical examination seemed to confirm the impression gained from the history that her cardiac and general physical status had not recently changed.

I then asked her how she had been affected by the flood. Tearlessly and still smiling, she slowly developed the story that she had lived alone in her own little home, but that it, with all her possessions, had been carried away by the flood. It became apparent, as she spoke, that this otherwise properly oriented woman had not fully assimilated the meaning of the enormous tragedy which was hers. Her mien and quiet, unspontaneous manner reflected her effort to hide the implications of the catastrophe from herself. Her complaint of weakness of the legs, however, clearly told of her burden through the one medium of expression available to her then. Treatment consisted simply in providing this woman with the special care she requested and an opportunity to work out her future plans with the help of a social worker.

Case 2. A young man of 26 years of age who had patiently awaited his turn, when finally asked about his problem, said he would like a prescription for athlete's foot. He slowly took off his shoes and socks. Between several toes was a typical, mild, chronic epidermophytosis. He stated that he had had the condition for several years and had never previously asked for treatment. In the situation of a busy dispensary crowded with many seriously ill patients, his minor complaint seemed out of place, and indeed the suspicion arose that he was simply using it as an

opportunity to get free medical advice. However, as the prescription was being written, he was asked a few general questions about himself.

From his wistful manner and the dependent way in which he related himself to the physician, it became clear that this young man felt lost and alone in the vast impersonal flood relief operation. He had actually come to the dispensary to get a few minutes of the kind of attention which he knew he would get from a doctor. He came for sustenance to stave off the feelings of loneliness and anxiety which were beginning to well up within him. The physician, aware of the patient's real reason for coming to the dispensary, was able, simply through his attitude and the quality of his attentiveness, to meet some of the patient's need within the time it took to prescribe and to outline treatment for the athlete's foot.

Case 3. A girl of 9, quiet, pale, and immobile, was seated between her parents. As the doctor approached, she turned her head sideways. The parents stated that she was suffering from "sinus" headaches. A general physical examination including transillumination was negative. She was not anemic. Her parents spontaneously went on to say that three months earlier a doctor had diagnosed an acute sinus infection.

In reply to direct questioning, they reported that since that time the girl had been free of headaches until the family was evacuated from their home three days previously, at which time the "sinus" returned. The child had not had a recent cold or fever. The parents, during the questioning, peculiarly continued to assume that the girl was suffering from a sinus infection and that the headaches were unquestionably on that basis. It was as though they could not talk about anything else and had to believe that "sinus" was responsible for their girl's trouble.

They were induced, however, to talk about other matters and finally mentioned that since the flood two siblings of the patient were unaccounted for. With an expression of dawning insight, the mother stated that their girl had been much troubled about her siblings but that they had asked her not to talk about this matter. The mother herself then suggested that perhaps the headaches were caused by worry about the siblings. The physician agreed with the mother, encouraged the parents to allow the girl to talk freely, suggested that they take her to the park for active play, and referred them to the Red Cross for help in locating the other children. The parents left cheerfully and considerably relieved.

Case 4. A woman who accompanied her 10-year-old niece stated that the child was suffering from upper abdominal pain. The girl herself sat quietly with her hands folded as the aunt listed the symptoms. Examination was negative, and in particular there seemed to be no basis to substantiate the diagnosis of gastroenteritis or other infectious or organic disease. The aunt was then further questioned about the girl's circumstances, and it turned out that the patient and her five siblings were living with their parents in a damp, crowded basement. The family had hurriedly left their flooded home, leaving behind all of their nicer clothing and personal possessions. The patient had not only lost these items, but had also lost a teddy bear of which she had been extremely fond.

In addition to living in an unpleasant small enclosure, the children were forced to remain heroically quiet and well-behaved in order not to jeopardize the family's place in the home where they were now living. As the aunt related the story, particularly about the loss of the teddy bear, tears came to the girl's eyes. It was clear that the child's loss, and the necessity to control her feelings, were responsible for her stomach pain. Simply bringing out the above facts in the course of understanding the problem and permitting the child to cry, served in this instance to relieve her pain.

Case 5. A 24-year-old man was seen rolling from side to side on a cot, holding his hands to his abdomen and groaning. When asked what his problem was, he replied, "My stomach." General physical examination was negative. After the examination he was asked, with firm conviction, "What is *really* bothering you?" He promptly replied, "I can't reach my family."

He told a story of working 48 hours on the dikes, of collapsing from fatigue, resting an hour, and then working 24 hours more. He told of his concern that he couldn't reach his family to find out how they were. Almost immediately after beginning to talk, his abdominal pain disappeared, and after 10 more minutes he asked for some food although he previously had been unable to eat.

DISCUSSION

The author was aware, before he undertook residency training in psychiatry, that many of his patients were under psychological stress of one kind or another. Like every other general practitioner, he had some conception of the financial, social, or family difficulties of his patients, but it was difficult for him to ask directly about such matters and investigate them as openly as he examined an infected finger or surgical wound. He felt that to probe into "personal" matters on the assumption that these contributed to fatigue or abdominal discomfort (implying, of course, that psychological factors were involved) was, compared with concern about purely physical matters, a relatively forbidden territory. Such questioning threatened to evoke the hostility of the patient who might interpret it as a violation of his privacy.

But the physician's fear of the patient's response in

these matters is like the hesitation of the medical student in doing a rectal or vaginal examination. With experience and knowledge comes the conviction that such "probing"—if done gently—is necessary. In addition, the patient can better co-operate with the procedure if the doctor feels comfortable about doing it. With psychiatric experience, the physician becomes increasingly comfortable in dealing with the thoughts, wishes, fears, and fantasies of his patients, as he becomes convinced of their importance. (See Case 5—"What is *really* bothering you?")

In the course of psychiatric training the physician becomes increasingly aware of the diagnostic importance of attitudes, postures, facial expressions, mannerisms, ways of speaking, etc. (See Case 1—the troubled forehead, the fixed smile, and the unspontaneous manner.) Likewise, he comes to understand that inappropriate behavior may be a significant clue to the psychological meaning of the symptom for the patient. Thus, the patient who calls the physician at night for an apparently minor ailment or, as in Case 2, the young man who came to a busy dispensary crowded with acutely ill patients, asking for treatment of a chronic athlete's foot, may be indirectly indicating what the treatment should be. What is it that the patient really wants? Why was it that the little girl with headaches (Case 3) turned her head away from the physician? It seemed probable to the physician that she felt guilty because part of her wanted to have her parents to herself, without the competition of siblings. In the cases cited, the patients presented the kinds of complaints the busy doctor sees daily, and the little signs mentioned furnished the clues for diagnostic formulation.

Many physicians find it difficult to observe behavior as objectively and with the same diagnostic interest as they observe other medical phenomena. Particularly irritating are the patients who depreciate the physician, refuse to have surgery or to follow directions, who move from one doctor to another, who don't appear for appointments, or who don't get better but keep coming back with the same or with more involved or elusive complaints. These and other behavior manifestations are symptoms which are just as significant in the diagnosis as a skin rash or leukocytosis.

It is true that the physician himself may have entered into the elaboration of a symptom by unprofessional conduct or by unwittingly stirring up fear, dependency, or other feelings in the patient; nonetheless, whatever the patient says or does is still a manifestation, a sign or symptom, of his illness, to the extent that his own personality is reflected in the illness. Whether or not the matter in question can be handled by talking about it with the patient, as it often can be, the doctor is not so likely to be irritated by such behavior if he is able to look upon it as a symptom rather than as a personal insult.

The roots of the patient's response to the doctor undoubtedly lie, in part, in hostile or affectionate feelings which existed much earlier in life toward the father or other important figures and which are now transferred onto the person of the physician. The ramifications of the subject are beyond the scope of this paper, but the point of the matter is that the physician can maintain a certain degree of objectivity and comfort by observing all behavior—sympathetically, to be sure—in the same light as all other medical phenomena.

These general observations suggest certain key questions which the practicing physician may find useful in orienting himself to psychological factors in the patient's illness. First, why did the patient really come to the doctor? Granted that the presenting complaint should have medical attention, or that the patient is uncomfortable, what is it that he really wants from the physician? Translating what the patient says into what he means is one of the major skills of psychiatry and related disciplines. It is a skill possessed in varying degrees, of course, by all students of human nature, but it can be developed by observation and training, and it is a skill which can be extremely useful in general medicine. The key question may be phrased in other ways: "Why did the patient really come to me at this time?" "What is he actually trying to say to me?" "Are his complaints covering up concern about something else? If so, what is it?"

Second, as the patient speaks of his illness, are his attitudes and feelings appropriate to the seriousness of the complaint? Does he seem overly concerned or too little concerned? Does he seem odd, peculiar, or even bizarre? Does he take delight in repeatedly describing his troubles? Does he volunteer that he wants surgery before the physician has even considered such a procedure?

Most patients should give a straightforward story, and their feelings and mood should be reasonably appropriate to the complaint. The neurotic or psychotic patient will probably behave otherwise. A vague or incoherent recital of symptoms may mean that the physical complaints are a front for deeper emotional problems. The patient with an early or mild depression may seem more pessimistic or be more preoccupied with his physical complaints than is warranted by their seriousness. The early or incipient schizophrenic may make a peculiar request of the doctor, as for example a young man in the author's general practice who, in a deeply troubled mood, asked that the doctor remove (normal) "bumps" from his forehead. Many schizophrenic patients request or attempt castration, while others attempt to secure castration symbolically

by seeking surgery on the face, nose, or teeth. Neurotic patients may request surgery for other reasons, at the same time treating the matter quite lightly, or becoming angry if the doctor hesitates. The attitude and mood of the patient, therefore, may be as diagnostic as other findings, and if inappropriate should give the physician pause. Unfortunately, surgery or even prolonged preoccupation with physical aspects of illness may aggravate the underlying emotional problem.

Third, what is the patient's response to the following remark: "It occurs to me that something may be troubling you, which you haven't yet mentioned." This question is useful when the physician suspects that emotional difficulties underlie the presenting complaint. Some patients are grateful at such an opportunity to talk, or may suddenly begin to cry. This reaction clearly confirms the doctor's suspicion, whether or not the patient goes on to explain why he is crying. Another patient may too vigorously deny that anything else is on his mind. The vigor of his denial likewise confirms the doctor's supposition. If the patient is able to mention other troubles, these may range, of course, from an unexpressed but realistic fear of cancer to the unrealistic fear of poverty which is so characteristic of the melancholic. If the physician has treated other members of the family, he is in a strategic position to guess at or to suggest what might be troubling the patient.

The above questions are a few which may help the doctor find the patient behind the presenting complaint. They supplement but do not take the place of a careful physical examination and necessary laboratory studies. The point of these questions is that to leave out entirely a psychological examination is like making a diagnosis on the basis of the history alone. The questions do not provide the basis for a thorough psychological examination, but they do provide a kind of psychological screening which is possible in a busy practice, and they do orient the physician to psychological factors in the majority of his patients and, in general, make the total examination far more complete.

CONCLUSION

The author, formerly a general practitioner, has commented on lessons learned during the study of psychiatry which he found useful when he temporarily returned to general practice. An effort was made to relate these lessons to actual case material. Questions have been suggested which may be useful to busy physicians in their efforts to visualize the larger problems behind the presenting complaint. Not included in this paper is the author's self-searching, which accompanied the care of these patients. How did he handle similar problems before he had had additional psychiatric experience? Some of the self-searching is reflected in the discussion, but as to the rest the author can only suggest, individual differences being what they are, that the reader conduct his own self-searching. Also not included in this paper is any reference to what other physicians have discovered for themselves along the lines of the topics discussed in this paper. It is basically a testimonial as to what intensive psychiatric training added to the day-to-day professional armamentarium of one general practitioner.

Historical Material Needed

In preparation for the observance of The Kansas Medical Society's centennial anniversary, members of the Committee on History are attempting to collect all material of historical interest. Physicians who can contribute information, records, etc., are urged to send such to

Committee on History
Kansas Medical Society
315 West 4th Street
Topeka, Kansas

Congenital Absence of the Vagina: Review and Case Report

Stanley L. VanderVelde, M.D., and Joseph J. Hovorka, M.D.
Emporia, Kansas

Congenital absence of the vagina, although not a rarity to the literature, is unusual in the ordinary physician's practice. The first case reported in the literature was by Realdus Columbus in 1572.[4] Engstadt[4] stated that vaginal aplasia occurs once in every 5,000 female births. Bryan et al.,[2] in reporting a series of 100 cases, found an incidence of one in every 4,000 female patients. Owens[15] found only six cases on 502,081 hospital admissions over a 34-year period at Charity Hospital in New Orleans.

One has only to talk a short time with one of these unfortunate patients to realize the mental state that results when these females find that they lack the necessary anatomy for happy and successful married life. Psychological reactions range from assumed complete indifference to severely depressed mental states with, in some cases, suicidal tendencies. All of these individuals possess a profound feeling of inferiority which over a period of time can produce complete emotional instability.

HISTORY OF TREATMENT

Attempts at correction of this malady go back many years. Depuytren[4] in 1817 is credited with the first attempt at construction of an artificial vagina. He separated the urethrorectal septum and inserted plugs, hoping for epithelialization. His results were disappointing. In 1872 Heppner[10] introduced skin flaps which later contracted. Thiersch grafts were attempted about this time but likewise failed. Gersuny,[10] in 1897, transplanted the anterior wall of the rectum with fair results. Sneguireff[10] transplanted the lower portion of the rectum into the vaginal opening, but again his procedure did not stand the test of time.

In 1904 Baldwin[1] suggested an operation and carried it out in 1907 to a successful conclusion. This consisted of isolating a loop of ileum and reestablishing intestinal continuity. The double loop of isolated ileum was then pulled down between the urethra and rectum. At a later date the double-barreled vagina was made into a single canal by applying a crushing clamp. Needless to say, in 1907 this required unusual skill. Daniel[10] reports a mortality of 17½ per cent in 79 recorded cases. This figure is doubtless low as many cases were probably not recorded in the literature.

Another procedure was devised about this time which carried a lower mortality. This was devised by Poppoff, Schubert and others.[10] It consisted of liberating the lower portion of the rectum and transplanting it into the urethrorectal septum, pulling the sigmoid down, and uniting it to the remaining portion of the rectum. Although the mortality with this procedure was less, disagreeable sequelae such as rectovaginal fistulae resulted fairly frequently.

Pedicle flaps were next to be used in the construction of a vagina. Jewett[10] in 1904, Graves[10] in 1921, Charbonnel[10] in 1921, and Fraenkel[10] in 1924 all used portions of skin in flaps from the hymen, fourchette, and vulva. These were united and invaginated into the preformed canal and held in place by packing or plugs. Annoying things such as hair growth in the canal and tendency toward atresia and obliteration resulted. The continuous wearing of an obturator was required to keep a patent vaginal vault.

Full thickness grafts raised from the adjacent inner aspect of the thigh were next used. This procedure was used by Frank[10] in 1925 with good success. The use of obturators was required for several months to maintain patency of the newly formed vaginal canal. This procedure required a three-stage operation consisting of at least eight weeks of hospitalization.

In an attempt to simplify the procedure, Thiersch grafts were revived. These free grafts were sewed to the surface of a preformed obturator and this was inserted into the urethrorectal septum. Results were uniformly good, and this procedure has carried through to the present day as the procedure of choice in the construction of an artificial vagina.

Another procedure utilized with fairly good results today merely requires the opening of the vaginal space and keeping it open with some substance to permit the tract established to epithelialize without the use of skin graft. This is similar to Dupuytren's original attempt in 1817. Either petrolatum gauze or iodoform gauze packs were originally employed in conjunction with obturators of paraffin or balsa wood covered with a condom. From 1935 to 1940 Wells, Kanter and Wharton[18] used these methods with uniformly good results. Biopsy from the vagina postoperatively showed squamous cell epithelium. Wharton suggested that the epithelium from the urogenital sinus forms a large part of the fetal vagina, growing around the müllerian ducts, and that probably in his cases the epithelium arose from the rudimentary vagina.

PRESENT DAY CONCEPT

The use of the Thiersch or split skin graft is probably the procedure of choice in construction of the vagina. Simple reconstruction without the use of skin graft, allowing the space to epithelialize about an obturator, also produces good results in many cases. Which procedure to use becomes a debatable question at the time of surgery. Counseller[7] suggests that in those cases where the vaginal tract can be opened easily and without bleeding from the venous plexus on each side, and where there is no difficulty in reflecting the peritoneum, the simple insertion of the obturator can be used without grafting. Where these requisites are not met, probably the safer procedure is the use of the Thiersch graft. Counseller[7] further stated that in one of his cases he encountered a bud thought to be a remnant of the müllerian duct. This was excised, and biopsy revealed it to be squamous epithelium.

Counseller[7] also stresses an important point that would apply to both procedures, namely the "contraction factor." McIndoe is responsible for this contribution. He stated that the "contraction factor" of any canal lined by a split graft may last for three to six months or longer and if the obturator or mold is removed before this "contraction" disappears, stricture of the new canal will ensue. Thus most failures to achieve good results are probably due to removal of the mold too soon. It should be removed only for cleansing purposes and for examination of the vaginal canal. Otherwise, it should be worn for at least six months or longer.

A discussion of construction of the vagina would not be complete without mentioning the work of Frank.[9] In 1935 Frank[9] examined a case of agenesis of the vagina where a deep dimple was found, apparently the result of vigorous attempts at coitus. The patient was instructed to apply pressure with glass cannulas regularly, starting with a small diameter and increasing gradually in size. In a few weeks a 7½ centimeter vagina was established which remained patent. Frank reports eight subsequent cases in which this method was used, all with good results. In another report, Frank[9] gives follow-up data on some of these cases for two or three years, reporting that the vaginal canal persisted in spite of lack of routine dilatation. In case of married patients, satisfactory intercourse was experienced by both partners.

SURGICAL TECHNIQUE

Regardless of what procedure is used, other than the pressure method of Frank, the opening of the rectovesical space is not difficult. A catheter or sound should be placed in the bladder. The anterior rectal wall should be palpated, usually with an assistant's finger in the rectum. The vaginal space is identified. Either a transverse or vertical incision is made over the vaginal dimple through the mucous membrane midway between the rectum and the urethra. A well defined plane of cleavage is encountered between the rectum and the urethra. By blunt separation the bladder is pushed upward and the rectum downward. The peritoneum is soon encountered and can be easily rolled upward from the anterior rectal wall by blunt dissection.

The amount of bleeding encountered is in direct proportion to the success of the subsequent take of the split graft. Many of these spaces can be opened with almost no bleeding. It is important that the space be dry when the graft or obturator is inserted. The obturator is inserted, covered either with split graft or condom, and left in place for two weeks. During this time the patient remains in bed, is kept on a low residue diet, and has a retention catheter in place. At the end of 14 days the obturator is removed; the canal is inspected and cleaned; the catheter is removed, and the patient is allowed to be ambulatory and soon discharged from the hospital with the obturator in place.

UROLOGICAL PROBLEMS

Numerous urological abnormalities are frequently found with vaginal agenesis. Phelan[16] et al. report that in a series of 72 patients with vaginal agenesis urinary studies were performed and 26 showed urographic evidence of abnormality. These abnormalities consisted of renal agenesis of either kidney, renal ectopia, unilateral fused kidney, and ureteropelvic obstruction. One case of a pelvic kidney was found at laparotomy. Such a condition could pose a real problem from a standpoint of possible trauma in any surgical procedure in construction of the vagina. The fairly frequent incidence of such problems makes it of signal importance for anyone contemplating construction of a vagina to have a thorough urological examination and study of the patient.

CASE REPORT

Mrs. I. B.,* a 22-year-old white female, was first seen at the age of 16 years at which time she presented herself to her family physician because she had never menstruated. She was told at this time that she had no vagina and no uterus. At age 22 the patient had been married two years and her husband had died of pneumonia. During her two years of married life no attempt at sexual intercourse had been made. After the death of her husband she decided something should be done to construct a vagina if this were possible.

Physical examination presented a normally devel-

* The original surgery on this patient was done by the late Clyde O. Merideth, M.D., Emporia, Kansas. The case report is taken from the files of St. Mary's Hospital, Emporia, Kansas.

oped, pretty, white female. Findings were not unusual with the exception of the pelvic abnormality. The external appearance of the vagina was normal. When the labia were separated, no vaginal entroitus was evident. A normal appearing mucous membrane appeared over the area where the vaginal entroitus would normally be. Bimanual rectal examination revealed no uterus but normal ovaries bilaterally.

Surgery consisted of incising the mucous membrane in a radial direction from a central point. Incisions were carried radially at 12, 3, 6, and 9 o'clock, respectively, carrying the incisions out to the supposed hymenal edge. By blunt dissection the vaginal opening was made between rectum and urethra for a distance of 8 cm. Minimal bleeding was encountered. Points of epithelium from the radial incisions were then sutured into the vaginal tunnel. A small glass tube 8 cm. long and 2½ cm. in diameter was placed in the tunnel and fixed there, holding the skin edges against the vaginal wall. A retention catheter was placed in the bladder.

The patient's convalescence was uneventful, and she was discharged from the hospital five days postoperatively. She wore the glass tube for 12 days, at which time it was removed. She performed daily manual dilatation of her vagina and was seen twice weekly for several months by her surgeon, who also did normal dilatation of the vagina. She does not remember how many months this treatment was continued but supposes it was about six months. After this period of time she had no dilatation other than sexual intercourse.

The patient was first seen by us on November 13, 1953, with findings of acute appendicitis. She was admitted to the hospital, and an appendectomy was performed. At this time, through the paramedian incision, the pelvis was examined. A vestige of a uterus approximately 1 cm. in diameter was found. Normal Fallopian tubes with fimbriated ends and normal ovaries were found. At this time pelvic examination revealed completely normal appearing external genitalia and vaginal entroitus. Three fingers could be inserted into the vagina with ease. Depth of the vaginal vault was 8 cm. Vaginal wall was normal appearing with normal appearing mucosa. A small amount of normal appearing lochia was present.

COMMENT

This case is of interest in that it gives a follow-up over the course of years. This patient had her vagina constructed at 22 years of age. After being married for 8 years and at 32 years of age, she states her sexual life is entirely satisfactory. Being an intelligent person, she has quizzed her husband about his satisfaction from their sexual intercourse and reports he has been completely content. This patient stated that she has told her husband she has no uterus but has never told him she had no vagina and had one constructed earlier in life. She also states that she is conscious of each menstrual cycle; her breasts become sore and although she has no discomfort she experiences symptoms of pelvic fullness for several days.

CONCLUSIONS

A discussion of congenital absence of the vagina is presented with emphasis on the various methods of surgical correction. Associated urological problems are briefly discussed.

A case of congenital absence of the vagina with subsequent vaginal construction without use of skin graft is presented. Follow-up of the case in married life is also presented.

BIBLIOGRAPHY

1. Baldwin, J. F.: The Baldwin operation for the formation of an artificial vagina, Surg., Gynec. & Obst. 569-570 (Oct.) 1927.
2. Bryan, A. L., et al.: One hundred cases of congenital absence of the vagina, Surg., Gynec. & Obst. 88:79-86 (Jan.) 1949.
3. Conway, H., and Stark, R. B.: Construction and reconstruction of the vagina, Surg., Gynec. & Obst. 97:573-578 (Nov.) 1953.
4. Counseller, V. S.: New surgical treatment for congenital absence and traumatic obliteration of the vagina, S. Clin. N. Am. 19:1047-1052 (Aug.) 1939.
5. ———: Congenital absence and traumatic obliteration of the vagina and its treatment with inlaying Thiersch grafts, Am. J. Obst. & Gynec. 36:632-638 (Oct.) 1939.
6. ———: Congenital absence of the vagina, J.A.M.A. 136:861-865 (March 27) 1948.
7. ——, and Sluder, F. S., Jr.: Treatment of congenital absence of the vagina, S. Clin. N. Am., 938-942 (Aug.) 1944.
8. Crossen, R. J.: Congenital absence of vagina, J. Missouri St. M. Assoc. 903-906 (Dec.) 1947.
9. Frank, R. T.: The formation of an artificial vagina without operation (intubation method), New York St. J. Med. 40:1669-1670 (Nov. 15) 1940.
10. ———: Evolution of the treatment for absent vagina, J. Mt. Sinai Hosp. 7:259-262 (Jan.-Feb.) 1941.
11. Kanter, A. E.: Congenital absence of the vagina; a simplified operation with the report of one case, Am. J. Surg. 30:314-316 (Nov.) 1935.
12. McIndoe, A.: The treatment of congenital absence and obliterative conditions of the vagina, Brit. J. Plastic Surg. 2:254-267, 1950.
13. Miller, C. J.: The Baldwin operation for aplasia of vagina, Am. J. Surg. 6:276-281 (March) 1929.
14. Owens, N.: A suggested pyrex form for support of skin grafts in the construction of an artificial vagina, Plastic & Reconstruct. Surg. 1946, 1:350-358 (Nov.) 1946.
15. Owens, N.: Simplified method for formation of an artificial vagina by split skin graft, Surgery 1942, 12:139-150 (July) 1942.
16. Phelan, J. T., et al.: Deformities of the urinary tract with congenital absence of the vagina, Surg., Gynec. & Obst. 97:1-3 (July) 1953.
17. Whitacre, F. E., and Alden, R. H.: Changes in squamous epithelium following the surgical treatment of absence of the vagina, Ann. Surg. 133:814-818 (June) 1951.
18. Wharton, L. R.: Further experiences in construction of the vagina, Ann. Surg. 1940, 111:1010-1020 (June) 1940.
19. ———: An improvement in the technique of constructing the vagina, Am. J. Obst. & Gynec. 60:871-874 (Oct.) 1950.

Faith marches at the head of the army of progress. It is found beside the most refined life, the freest government, the profoundest philosophy, the noblest poetry, the purest humanity.

—T. T. Munger

The Widening Scope of the State Hospital, with Special Reference to Geriatric Medicine and Surgery

C. Maxwell-Joyner, M.D., and Rudolf Hofbauer, M.D.

Larned, Kansas Wichita, Kansas

During the past decade, as most of us know, a transformation has taken place in the care of our mentally sick. Stimulated by public opinion, the press, and their own convictions, authorities responsible for providing hospital care for the ever increasing numbers of mental patients have embarked on an impressive program to make these hospitals and this care ever more effective.

Trained psychiatrists, psychologists, social workers, special therapists, and other trained personnel are becoming more available for work in state hospitals. A veritable renaissance in the care of the mentally afflicted has thus taken place. Gone is the conception of the state hospital as a custodial institution only. Most mental hospitals are now alive with every activity conducive to the welfare and treatment of the mentally afflicted with programs directed toward their rapid rehabilitation and recovery.

Thus it has come about that a great expansion has taken place in the scope of the work done in state hospitals all over the country—in some hospitals to a greater degree than in others. Greater and wider responsibilities are being borne by them, and not the least of these is the care of physical infirmities from which mental patients, in common with other people, suffer. With increased life expectancy resulting from the discovery of antibiotics and impressive advances in medicine and surgery, more and more patients being admitted are elderly people who have thus been preserved but in whom the inevitable chronic disabilities of old age are present, such as arteriosclerosis, diabetes, hemiplegia, peripheral vascular disease, chronic heart and kidney conditions, and, in the surgical field, prostatism, hernia, fractures, uterine prolapse, hemorrhoids, and gangrene.

These physical disabilities have an important bearing on the mental condition of the patient; indeed, it may be stated that they are in certain cases the cause of the mental breakdown, as for example, chronic brain syndrome (arteriosclerosis). It is now being widely acknowledged that many senile infirmities with mental counterparts are conditioned by glandular failure, and many remarkable improvements in the mental state of patients have been recorded following the use of appropriate hormonal therapy.

In this connection we would like to mention the usefulness of the gonadotrophic hormones fortified with thiamin and glutamic acid in the case of male climacteric and senility, and fortified androgens in female senile cases. It is in this field, that of mental hospital practice, that our interests have been specially centered to see just how much can be done to improve the mental condition of the patient by alleviating or correcting any physical disease or disability.

Many psychoses, e.g. schizophrenia, cannot be explained on physical grounds and must be elucidated and treated by psychiatric and psychological methods. In many, psychoanalysis may well be the method of choice. It is now stated, however, that even schizophrenia is being stripped of its mysteriousness. Definite changes in the cellular chemistry of the brain cell of schizophrenics have been demonstrated and also changes in the blood (Houston Texas Research Center). Then we have general paresis, syphilis of the central nervous system, psychoses following surgery (prostatectomy, hip-pinning, hysterectomy, ovariotomy), childbirth, menopause, goiter, shock, carbon monoxide poisoning, etc.

We feel, therefore, here at Larned, that there is a big field for co-operation between the psychiatrist, the internist, surgeon, pathologist, biochemist, and psychologist. We are endeavoring to combine our resources and collaborate on each individual patient admitted to the hospital. While the patient is under the care of the medical and nursing staff, having the mental condition studied and worked up, a much more intensive physical survey is being conducted than was heretofore possible. The meshes of the net are being made smaller, and a great variety of pathological conditions, many of which have an important bearing on the patient's mental illness, are being found. A colleague of ours, the hospital radiologist, said he is surprised at the great number of pathological conditions he has observed in the roentgenograms he has studied and read.

It will be seen that a challenge and opportunity is presented to state hospital physicians who are working in the physician-surgeon section and whose duties embrace the fields of internal medicine, surgery, and pathology. The neuropsychiatrist simply has not the time to make adequate evaluation of the general physical condition of the patient. It is in many cases im-

possible to have investigation at outside clinics or hospitals for physical conditions in the mentally afflicted, for obvious reasons. Therefore, we consider it incumbent on the medical staff of the state hospital, helped by their consultants, to provide a service inferior in no way to that which could be obtained at a good hospital or clinic.

In the *Report of the California Department of Mental Hygiene,* 1950-1952, page 57, it is stated, "In order to provide good care, the mental hospitals *must* have the same facilities found in any general hospital, with a complete range of medical services to supplement the special facilities required to treat mental illness." . . . "The continual tensions, anxieties, frustration and resentment felt by the mentally ill patient show a heavy toll in *physical* as well as mental symptoms."

It has been said that, through the development of psychosomatic medicine, thanks to the researches of Cannon and others, we are coming to a realization that the emotions play a large part in the functioning of the various organs. W. Overholser in *Geriatric Medicine,* page 263 (W. B. Saunders, Philadelphia, 1944), states that there is a close relationship between the body and the mind. Metaphysics teaches that the mind is an epiphenomenon of the brain (my interpolation). In the past, internists and surgeons have been somewhat oblivious of this fact, and, in the same manner, the psychiatrist has erred in sometimes paying too little attention to some of the physical findings in his patients. Today, however, we find the psychiatrist, the internist, and the surgeon, as well as the various other specialists, meeting on a common ground and recognizing that they are dealing with a human being and that human being must be considered *in toto* rather than as an "assemblage of parts."

We feel that it is not sufficiently realized what a good medium the state hospital, with its generally large population, provides for research into chronic disease, medical and surgical as well as in neuropsychiatry, and we would like to draw our colleagues' attention to this aspect of state hospital practice. Many patients remain in state hospitals over prolonged periods, and this very fact gives a good opportunity to trace the course of chronic disease which may exist. Pathological processes can be followed, changes recorded, and followed through to autopsy or resolution. Definite contributions to geriatric medicine and surgery can thus be made, and this aspect of mental hospital practice should prove a stimulus to those doctors of the physician-surgeon group.

Not only is opportunity afforded for the sustained study of chronic illness, e.g. geriatrics, but screening programs for cancer, tuberculosis, and other diseases which have a definite public health value may well be undertaken by the medical staff. As an example of this type of work we would like to mention an investigation at present in progress at the Larned State Hospital into the incidence of cancer of the cervix uteri among the 500 aged female patients in this hospital. This investigation is a part of our general cancer program which has been in existence more than two years. It is based on the following:

1. Skin—Inspection
2. Oral—Inspection
3. Breasts—Palpation
4. Chest—Examination. X-rays, bronchoscopy, sputum examination for cells
5. Gastrointestinal—Rectal. X-rays, gastric brush method, washings, etc.
6. Uterus—Pelvic examination. Routine cervical cytology and biopsy
7. Prostate—Examination (rectal) with or without cytology.

This detection system is that advised by the Eugene Carey Cancer Foundation, Inc., 63 Wall Street, New York.

If there are abnormal findings, special investigations are then made to supplement this examination.

The following results from gynecological examinations made on 331 patients have been obtained. These female patients were not selected because of any suggestive symptoms, and were all presumably healthy.

	Number	*Percentage*
1. Cases showing positive cellular pathology for cancer of the cervix or endometrium, i.e., groups IIIB, IV and V (cytological grouping)	22	6.64
2. Cases showing suspicious cellular pathology, but where it is not believed that the patient has cancer of the cervix—group IIIA	40	12.08
3. Cases showing negative cellular pathology, i.e., groups I and II	269	81.26
4. Ring biopsies done to date on cases showing positive cellular pathology ..	14	
5. Ring biopsies positive for cancer of the cervix	4	
6. Positive ring biopsies in total number of positive cellular biopsies to date ..		28.57
7. Positive ring biopsies in total number of cases screened		1.20

Maurice Fremont Smith and Ruth M. Graham, Boston, in an article in the *Journal of the American Medical Association,* Volume 150, Number 6, page 587, state, "Among 704 consecutive new women patients coming to the office of an internist, pelvic cancer was detected by vaginal smear in 10 cases (1.4 per cent). We feel strongly that vaginal smear sampling is not enough. A scrape of the squamocolumnar area of the cervix in addition to the vaginal smear is necessary, as the vaginal smear collects dead cells that have been desquamated from the growth and are lying free in the vagina or at the cervical os for some time. The earliest changes, according to Ayre, in the

malignant or possible pre-malignant state of the cells can best be found thus. This was also pointed out by Professor Hofbauer of Cincinnati in an article in the *American Journal of Obstetrics and Gynecology* as early as 1934."

We quote the above as an example of the type of investigative work that may profitably be done in state hospitals. When we consider that over 50 per cent of the hospital beds in this country are occupied by mental cases, we come to realize what a large pool of patients is available for such or similar investigations. We do not believe that any harm can accrue to the patient's mental condition if these investigations are conducted carefully and with a view to avoiding psychic trauma. Many other forms of examinations, e.g. mass radiography for tuberculosis, laboratory procedures, and courses of anti-syphilitic treatment are done in state hospitals so that it would not seem that special examination involved in cancer screening or other similar observations could do psychic harm. We tell the patients that we are just giving them a "check-up."

I trust that the foregoing remarks and observations outline how the increasing volume of organic disease requiring treatment in state hospitals presents a challenge to the medical, nursing, and laboratory staffs. I consider that this challenge can be met by ever increasing clinical zeal in the application of modern methods to the physical care of the mentally afflicted and incidentally, perhaps, contributing to the advancement of scientific medicine in our mental institutions.

I wish to thank Dr. J. T. Naramore, medical superintendent, for permission to publish this article.

The World of Books

Is the most remarkable creation of man.
Nothing else that he builds really lasts;
Monuments fall,
Nations perish,
Civilizations grow old and die out
And after an era of darkness
New races build others.
But in the world of books are volumes
That have seen this happen again and again
And yet live on, still young,
Still as fresh as the day they were written,
Still telling men's hearts
Of the hearts of men centuries dead.

—*Clarence Day*

PRESIDENT'S PAGE

Dear Doctor:

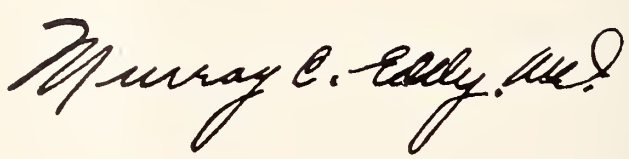
This is your and my Thanksgiving letter. While it states nothing new, it is intended to give some cause for reflection and perhaps some individual re-evaluation.

The philosophy that complete regulation of thought and action by government is necessary has already been imposed upon great segments of the world population. We can be thankful that we are free to live our lives, make our friendships, and practice our profession without political or governmental interference.

Men of medicine have always been leaders in the continued struggle to maintain the dignity of the individual and to perpetuate good and honest government for the people. Dr. Benjamin Rush, Dr. Lymon Hall, and Dr. Carter Broxton were three of the signers of the Declaration of Independence. Dr. Charles Robinson was the first governor of our state, Dr. J. P. Root our first lieutenant governor, and Dr. J. W. Robinson our first secretary of state. These men were leaders in the incorporation of your state Society.

It is my hope that medical men everywhere and particularly those in Kansas will work honestly and diligently to keep our heritage bright.

Yours,



EDITORIAL COMMENT

Is It Art?

During the reign of Joe Louis as heavyweight champion, a sportswriter was criticized for having referred to one of his fights as an exhibition. The columnist replied that the popular concept of this word is a situation where an artist places something upon canvas for the public to look at. Then you begin to wonder, is it art?

Not that the question is vital to a prize fighter, nor even to a painter, and perhaps not at all to a physician, but here too the question recurs. Is medicine an art or is it business and, if a combination of both, can a definition separate the two?

Perhaps not. Perhaps the subject should not be explored at all because the answer is something less than we hope to hear. And yet, among the ashes of misinformation and among the rubble left by those to whom no intangible has meaning, may yet be found a sparkling fragment that is worth looking at.

Even the basest among materialists will recognize that a single act may be performed more perfectly by one man than by another. The sheer selfish joy of having performed to the limit of one's ability brings pleasure, and if that expression of his best whether as a bricklayer or a sculptor is not art, then it is something else synonymous with art.

In days gone by when the helpless doctor sat in that strangely silent family circle holding the dying patient's hand, then medicine was an art. Today he confidently prescribes a drug and leaves. And that is business?

The artist is a perfectionist. He always strives for and never quite achieves the masterpiece. One success merely spurs his desire for improvement whether he is a prize fighter or a physician. He is an artist if he has an ideal that is greater than can be reached. He is an artist if he loves his work and in each effort senses the satisfaction of a new creation. He is an artist who would not change his field regardless of fame or power or wealth. He is an artist who believes that what he does is true.

There can never be a question about this in the artist's mind. The physician has an answer on whether medicine is an art, but when he comes in contact with the unskilled person misunderstandings arise. It is at the exhibition that the work is criticized and condemned. So art is an individual and a personal accomplishment. Call it what you will, after this touches the public, the consumer, something happens. He cannot come in contact with an art and leave it as it was.

To the consumer it is business. He gives his time to view an exhibition. He purchases a ticket to a prize fight, he employs a physician, and the art is now related to his own particular sense of values. It becomes his property in goods or service, and he places his own evaluation upon it.

So actually medicine is both, art to the physician and business to the consumer. This gulf can never be completely erased, and a certain degree of conflict must always be present just as it has been for all forms of artists through the ages, who offered the objects of their skill for public use.

Federal Aid in Kansas

PART II

Among the 35 different federal grant-in-aid programs operating in Kansas, six are in the field of agriculture, although one of these could be considered also under another heading. The story of these grants is here published for informational purposes, and no attempt is made to evaluate the services performed. Some additional information on the subjects discussed in this column is available and may be obtained by writing the JOURNAL.

Agricultural Extension Services funds are administered in Kansas through Kansas State College at Manhattan. On a federal level this project has been operative in the Department of Agriculture since 1914. Money is available "to aid in diffusing among the people of the United States useful and practical information on subjects relating to agriculture and home economics, and to encourage the application of the same."

A total federal grant of almost 32 million dollars was available in 1953, allotted to the different states about as follows: 4 per cent was given on the basis of need, and the remainder was divided on two formulas, one on the basis of rural population and the other on farm population.

Kansas has used federal aid in this field since the legislature passed a resolution for its acceptance on March 1, 1915. Expenditures for agricultural extension work have increased rather regularly to the extent that there has been an increase of one million dollars within the last four years, mostly from local sources.

In 1953 the federal government gave $665,340, the state contributed $407,851, and the different counties appropriated $1,455,863, making a total of $2,529,054. Considering only the federal portion of this grant, Kansas received 2.27 per cent of the total spent in all states.

Agricultural Experiment Stations represent the second federal aid program in this category. This be-

gan in 1887 and has continued to the present time. Even though only a small portion of this grant requires matching, Kansas still gives more than twice the federal amount. In 1953 this was $237,127 from the United States and $583,604 from Kansas, making a total of $820,731. Nothing is shown as coming from county governments. Again, for its possible interest, Kansas received 1.99 per cent of the total federal grant or something under the average for all states.

Cooperative Farm Forestry, the third program, is a small activity in which Kansas paid $3,651 and the federal government granted $1,620 in 1953 for a total of $5,271. This too is administered through Kansas State College and serves to assist farmers in establishing windbreaks, shelter belts, etc. It has been operative in Kansas since 1935. The federal grant is more than 10 million dollars, so Kansas receives only 0.01 per cent of the total, a negligible sum when compared to participation elsewhere.

The fourth, also administered by Kansas State College, is for Agriculture and Mechanic Arts. This has been received in Kansas since 1891 and last year amounted to $88,586, all from federal sources. The total United States program cost in excess of five million dollars, so the Kansas share was 1.76 per cent. This is an outright grant with no matched funds to pay salaries for administrative officials and faculty in the schools of agriculture and mechanics.

The Kansas State Board of Agriculture administers one program for which federal aid is received. This is listed under the title of Research and Marketing and is described as a study of "ways and means of improving storage and transportation facilities, developing new uses for farm products, and expanding marketing outlets." The program has operated since 1948.

Federal grants must be matched dollar for dollar. In 1953 the federal sum was $27,099.59 and the state sum exactly the same to make a total of $54,199.18. Kansas receives 2.3 per cent of the federal appropriation.

The final project is Distribution of Surplus and Other Food Commodities, which operates under the Kansas State Board of Social Welfare. In 1933 Kansas received its first emergency benefit, and in 1935 the project began on a permanent basis. The operation of this program affects several agencies, charitable hospitals, schools, and others, but it was listed under agriculture because its principal purpose is "to prevent the waste of food commodities and to support the price of certain agricultural products."

The program appears to be growing in cost with federal participation assuming almost all the expense. In 1947 the federal government gave $227,222, last year $1,063,385. Kansas contributed $23,817, making the project come to $1,087,202. There is no county charge except for freight, storage, and preparation of foods which have not been listed. The primary federal regulation is that these foods will be properly disbursed and "used in addition to, and not in substitution for, commodities normally purchased." The total United States expenditure was $60,140,000, of which Kansas received 0.96 per cent.

A summary of these six programs under agriculture is as follows: counties spent $1,380,620, the state $936,925, totalling $2,317,545. In addition, federal grants received in Kansas came to $1,660,486 out of a total expenditure of $120,270,000, or 1.38 per cent of these federal appropriations were spent in this state.

Next month an analysis of federal aid to education will be made.

Annual Meeting in Hutchinson

Those who regularly attend annual sessions of The Kansas Medical Society are aware of the many opportunities afforded at the meeting. The next meeting will be held at Hutchinson, May 1-5, 1955, and promises to be one of the finest.

Adequate housing facilities are available in hotels and motels. Those desiring to choose a particular place to stay might write the Chamber of Commerce, Hutchinson, at an early date.

The program committee has completed arrangements for the scientific session. Eleven outstanding speakers from various sections of the country have consented to present papers for a well-rounded program. Like last year, the guest speakers will comprise a panel for afternoon sessions in which all physicians are cordially invited to participate.

Commercial and scientific exhibits as well as the scientific program will be presented at the new arena in Hutchinson where adequate parking space and lunch facilities are available. Buses running between the downtown district and the arena will be available for those without transportation.

Various committees arranging the meeting have already spent many hours preparing this outstanding event. The Committee on Scientific Exhibits is making a special effort to have a wide variety of displays. Space is no problem in the large arena, and a record number of exhibits is the committee's goal. Members of the Society are welcome to prepare exhibits for presentation at the meeting. Recognition will be given outstanding contributions.

The House of Delegates will meet at the usual times. Golf and skeet events will be held on Monday, as in the past. The beautiful ballroom in the Baker Hotel will be the scene of the annual banquet customarily held on Wednesday evening.

Members of The Kansas Medical Society will hear

more of these and other events of the meeting in future issues of the JOURNAL and in correspondence from the Executive Office. This early report is presented to remind the membership of the dates, to encourage attendance, and to assure a fine meeting next May.

THE MONTH IN WASHINGTON

Editor's Note. The following summary of Washington news was prepared by the Washington office of the A.M.A. for distribution to state and regional medical journals.

Although the elections back home are more stimulating than Washington doings these fall weeks, some of the quiet planning going on at the Pentagon should be of more than passing interest to physicians, young and old. The objectives are familiar: first, to insure a steady supply of physicians for the services; second, to improve the medical care program for military dependents. Primarily responsible for working things out are Dr. Frank Berry, Assistant Secretary of Defense for medical affairs, and the officers assisting him.

To insure that the services will get the physicians they need after the scheduled expiration of the Doctor Draft Act next July 1—without disrupting residency training—a plan bearing the formidable name of the Armed Forces Reserve Medical Officer Commissioning and Residency Consideration Program has been put into effect. It applies only to interns who have had no prior military service and who therefore have a two-year obligation for service under the regular draft.

The plan's first deadline was October 10. By that time these young physicians were to have sent in to the Defense Department a form with the following information: Their first, second, and third choices among the services, whether they wanted additional deferment for residencies and if so choice of hospitals, and the preferred specialties. Any in this group who do not apply for reserve commissions will be subject to the regular draft, will not be considered for residency deferments, and will not have a choice of services.

There is another problem involved. It is estimated that about half of the interns will want residency deferments. However, not more than a quarter can be deferred if the Army, Navy, and Air Force are to get their quotas of physicians. This is being resolved by a lottery. Those winning deferments will stay in the reserves and be called up for duty as their specialties are needed after the completion of their residencies. Those losing out will be called as needed at the end of their internships. The 50 per cent not asking for deferments will be allowed a choice of the month to be called up, a privilege not accorded the others.

On the dependent medical care program, Dr. Berry's annual report discloses that the department is all set to put the expanded plan into operation, should Congress enact it. An implementing directive has been drawn up, a tentative fee scheduled modeled on the VA "Guide for Medical Services" has been prepared, and a uniform "Military Dependent Identification Card" has been developed and placed in limited use by the Navy and Air Force.

A dependent care bill was introduced last session but not pressed by the Defense Department. It provides a uniform program for the three services, with dependents defined and the extent of care limited. It also would have the military medical departments take care of all the dependents they could handle, with only the remainder going to private physicians and hospitals. The American Medical Association believes this should be reversed, with emphasis on private, non-government care for dependents.

The Defense Department is interested in other devices to keep up the quality as well as the number of its physicians. One of these is a scholarship program, which would require one year of military service for each scholarship year. Because regular draft time could be served out this way, any scholarship contract would call for a minimum of three years' active duty. The department has high hopes that this program will be authorized by the next Congress. It also is hopeful that, once in operation, the scholarship contracts would result in more young physicians joining the regular Army.

Meanwhile the Hoover Commission on Organization of the Executive Branch and the Kestnbaum Commission on Intergovernmental Relations continue with their studies and report-writing, efforts that now are definitely unspectacular but that ultimately could mean important changes in the government's medical programs.

The Hoover Medical Task Force is nearing the end of its long review of all federal medical operations. Its recommendations will be submitted to the full commission for consideration in the commission's report to the President, due at the White House next May.

The Kestnbaum commission's work of greatest medical interest is the study of health grants-in-aid, on which a special committee has just completed its report. The full commission is scheduled to report back to Congress by next March.

Clinicopathological Conference

Case Presentation

Dr. William E. Larsen, resident in medicine: This 66-year-old Negro was admitted to KUMC on August 10, 1953, and died August 22, 1953.

The chief complaints were cough, shortness of breath, and weakness.

The patient was in good health until August, 1952, at which time he experienced a sudden onset of dizziness and transient blindness accompanied by numbness and weakness of the left arm and left leg of ten days' duration. In December, 1952, he had an episode of numbness and weakness of the right arm and right leg. He also found that although he could see he was unable to read, and he noted some impairment of his memory. Following the episode in December, 1952, the patient quit his work as a saloonkeeper. Strength gradually returned to his right arm and both lower extremities. The left arm weakness persisted.

Three months prior to admission the patient noticed the onset of a nonproductive cough, first with effort and then at rest while lying in bed at night. With the nocturnal coughing he became dyspneic. He would sit up in bed until the cough was relieved. The cough became productive of a whitish mucoid sputum. These symptoms gradually progressed until his KUMC admission in August, 1953.

Past history revealed that he had the usual childhood diseases plus typhoid fever and malaria. He passed a large tapeworm when he was a child. He had gonorrhea at 18 years of age with five subsequent episodes. He also stated that he had a prolonged diarrhea and abdominal pain beginning about six years prior to admission and lasting for about four years. This was accompanied by red blood and black tarry stools on several occasions.

Significant family history revealed that one brother and one sister died with pulmonary tuberculosis. His stepson died of tuberculosis in February, 1953.

The system review was significant in that he had a nonproductive cough of about three months' duration and he had nocturia once nightly of several years duration. Weight loss of 20 pounds occurred during the year prior to admission.

On physical examination, the well developed, poorly nourished Negro male of 66 years of age appeared to be in respiratory distress while lying in bed with head elevated. Conjunctivae were pale. The pupils were of average size and regular. They reacted sluggishly to light. The optic disc margins were distinct. There was an old scar in the left fundus but no hemorrhages were seen. The retinal arterioles were narrowed. The mouth was edentulous, and there was a diminished gag reflex. There was no lymphadenopathy in the neck or supraclavicular region.

The trachea was slightly deviated to the right. Rales of a fine moist nature were heard scattered in the left anterior infraclavicular region and across both posterior lung fields at the bases. The heart was of normal size with good quality tones. No murmurs were heard.

The abdomen was soft, and no masses or organs were palpated. The prostate was of normal size and consistency. There were no abnormalities of bones or joints.

Deep tendon reflexes were increased on the right. The left arm was ataxic. Bilateral ankle clonus was elicited. Vibratory sensation was absent in both lower extremities. The pulse rate was regular at 80 per minute. Brachial blood pressure was 110 systolic, 65 diastolic.

On laboratory examination, the urine specific gravity was 1.020 with two plus albumin. The urine contained 3 to 5 pus cells per high power field with some waxy, hyaline and granular casts. Red blood count was 4.7 million with 14 grams of hemoglobin. White blood count was 19,550 with 82 per cent polys, 11 per cent lymphocytes, 5 per cent monocytes, 1 per cent eosinophiles, and 1 per cent basophiles. VDRL was negative. The blood non-protein nitrogen was 57 mgm. per 100 ml., creatinine 2.1 mgm., sugar was 98 mgm., and the serum sodium was 136 mEq./L. The total protein was 7.1 grams per 100 ml. with 5 grams of albumin and 2.1 grams of globulin. The total serum bilirubin was 1.0 mgm. per 100 ml. The sedimentation rate was 5 mm. in one hour. Electrocardiogram and x-rays will be demonstrated.

A few hours after admission to the hospital and while being bathed in the bath tub, the patient became dyspneic and cyanotic. He was returned to his bed where examination revealed no change from the above listed findings. With oxygen by mask he became comfortable. The circulation time arm-to-tongue was 17 seconds, arm-to-lung was 11 seconds, and the venous pressure was 84 mm. saline. It was noted that he coughed frequently in paroxysms producing a whitish mucoid sputum. Several specimens of this sputum were sent to the laboratory for study. He complained of some right anterior chest pain which was controlled by aspirin and codeine.

While eating his lunch on the fifth hospital day, the patient aspirated a small amount of food fol-

From the University of Kansas Medical Center. Edited by Glen R. Shepherd, M.D., and Mahlon Delp, M.D., from recordings of the conference participated in by the departments of medicine, radiology, pathology and oncology and the third and fourth year medical students.

lowed by severe paroxysms of coughing for 20 minutes. Oxygen was necessary to relieve dyspnea and cyanosis. He was given procaine penicillin, 600,000 units daily. The white blood count on August 11 was 26,150 with no change in the differential. On August 14 it was 35,000, and by August 17 the white blood count was 54,000 with a differential of 88 per cent polys of which 76 were filamented and 12 were nonfilamented, 8 per cent lymphocytes, 1 per cent eosinophiles, 2 per cent monocytes, and 1 per cent metamyelocytes. Lung field examination revealed no changes in breath sounds.

By August 20 edema was noted on the upper right leg and bilaterally on the lower legs. Presacral edema was noted by August 22. He received a 0.6 mgm. dose of digitoxin IV at 12:00 noon. Three hours later the patient's dyspnea became worse and there was moderate cyanosis, although only a few rales were present over the posterior lung fields. The dyspnea remained the same and the pulse was strong at 100 per minute. At 4:05 p.m. the patient slumped over in bed and was pronounced dead.

Dr. Mahlon Delp, chairman: Any questions of Dr. Larsen?

Q: What were the results of skin tests?

Dr. Larsen: Positive tuberculin and positive histoplasmin; negative blastomycin and negative culture of urine.

Q: Did the patient have calf tenderness?

Dr. Larsen: This patient had no tenderness at any time during his hospitalization.

Q: What was the appearance of the sputum?

Dr. Larsen: The gross appearance was white and mucoid with a volume of about 30 to 60 cc. daily.

Q: Did he have difficulty raising that or did it come up easily?

Dr. Larsen: It came up easily.

Q: Was it gelatinous, opaque or clear?

Dr. Larsen: It was clear and watery.

Q: Was it sticky?

Dr. Larsen: Yes, it was sticky.

Q: Did an oil form on it? Was it allowed to collect for 12 to 24 hours?

Dr. Larsen: It did not layer.

Q: What was the temperature range?

Dr. Larsen: The patient was afebrile during his hospital course.

Q: Did this man smoke?

Dr. Larsen: I don't know.

Q: Had this man had any occupation other than that of saloon keeper?

Dr. Larsen: No, at least not within the 15 years preceding hospitalization.

Q: Did the patient give a history of ever having been treated for syphilis?

Dr. Larsen: No.

Q: Was there any examination for blood in the stools?

Dr. Larsen: No, that was not done.

Dr. Delp: Did you notice any blood in the stools?

Dr. Larsen: The stools were not tarry during hospitalization.

Q: What about the intensity of the breath sounds? Was there any instance of alteration in respiration?

Dr. Larsen: The breath sounds were fair to good over his entire chest. There were no signs of any obstruction.

Q: In spite of dyspnea and cyanosis?

Dr. Larsen: In spite of dyspnea and cyanosis.

Dr. Delp: Mr. Becker, let's see that electrocardiogram.

Mr. Donald E. Becker, fourth year student:* The rate is 110. There is regular sinus rhythm as evidenced by T waves in leads I, II, aVr.

The axis deviation is to the left about 5 to 10 degrees. All the T waves in these leads are upright. There is ST segment elevation in leads V_1, V_2, V_3 and aVf, but the elevation is only one millimeter, which is normal.

My interpretation is sinus tachycardia.

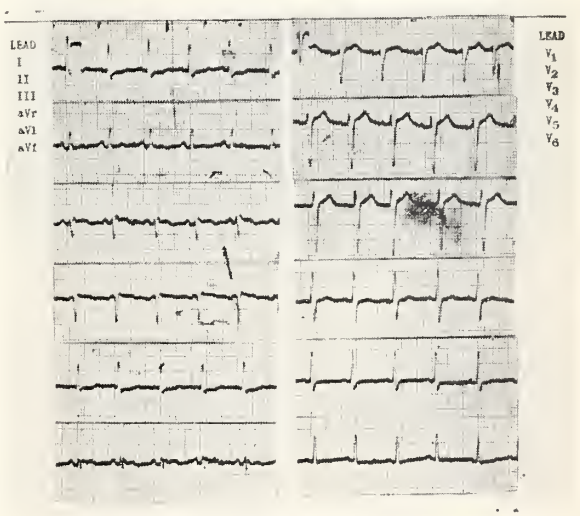


Figure 1. Electrocardiogram showing sinus tachycardia.

Dr. Delp: Do you have any comments, Dr. Lin?

Dr. T. K. Lin, medicine: It showed only tachycardia.

Dr. Delp: Mr. Wilcox, let's see the x-rays.

Mr. Robert N. Wilcox, fourth year student:* We have three chest films and one intravenous pyelogram, a skull film, a K. U. B. film and a gastrointestinal film showing the stomach and small bowel.

The first chest film was taken on July 16, the second

* Received M.D. degree in June, 1954.

on August 11, the third on August 15. About the only thing you can say about these films is that there is an extensive patchy infiltration in the lower lobes of both lungs. The apices are relatively clear. There are no hilar nodes seen. The heart is normal in size. The trachea is in the midline.

In the next film we see this diffuse patchy infiltration in the lower lobes of both lungs and again in the third film. The apices remain comparatively clear. The costophrenic angles are clear.

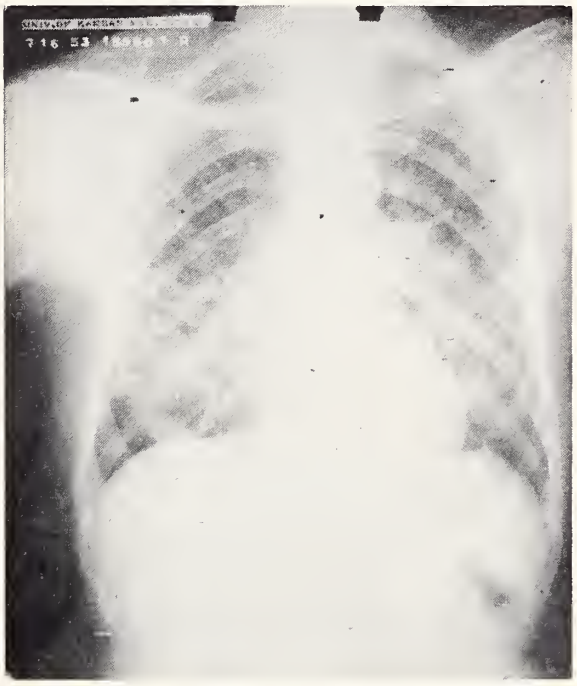


Figure 2. X-ray of chest showing extensive patchy infiltration in the parenchyma of the lower lobes.

I think our differential diagnosis from these chest films must include chronic passive congestion, pneumonia, with which this picture is compatible, sarcoidosis, and lymphatic spread of a malignancy.

We would have to consider a chronic passive congestion, but I think the normal size of the heart would rule that out. We were considering sarcoidosis but I think we would expect to see, though we would not necessarily have to see, an increase in hilar nodes. It was not noticed in these films. The last thing we must consider is a lymphatic spread of malignancy. We can't rule this out from these films. We certainly do not see malignancy in the films of the gastrointestinal tract. In the pictures of the stomach we see the normal rugal folds; the duodenal bulb has filled well. There are no abnormalities of the large or small intestine. I would consider this a normal film.

The skull film is normal. There may be some narrowing between C 5 and 6. There are no evidences of bone destruction.

The intravenous pyelogram was taken 45 minutes after injection of the dye. We can see here that the markings of the left kidney are absent. On the right side they are normal. I would consider this to be a non-functioning left kidney. Perhaps here we can see the prostatic shadow. There is no evidence of any bone destruction.

Dr. Delp: Dr. Hogan or Dr. Litton, have you any additional comments?

Dr. L. O. Litton,* radiology resident: I think Mr. Wilcox did very well.

Dr. Delp: First of all, this patient did have neurological signs and symptoms extending over a period of time. We should have some explanation for that.

Next, there is the family history of undoubted contact with pulmonary tuberculosis. That must have a bearing upon the discussion and require some comment. The patient did have dyspnea. Was the dyspnea cardiac in origin or was it perhaps cardiopulmonary —a combination of the two? I think we must discuss this.

Finally, of course we wonder why this man died in such difficult circumstances so soon after coming into the hospital. It is necessary to explain why he worsened so rapidly.

Mr. Coble, may we have your differential diagnosis?

DIFFERENTIAL DIAGNOSIS

Mr. Rollo J. Coble, fourth year student:† In this patient's previous illnesses are descriptions of two neurological deficit episodes. I think that the history of the sudden onset with improvement, the relatively short course of those episodes, and the fact that he had not had continuing increase in his symptoms are rather good indications that they were cerebrovascular accidents, probably hemorrhagic in nature. I don't feel that they have any bearing on this present illness.

The history of a four-year course of diarrhea with occasional bleeding I find very difficult to explain. He had been asymptomatic in that respect for three years. And again, I don't think that had any bearing on the present illness. I think that the man probably had a bleeding ulcer at that time. The possibility of a gastrointestinal malignancy is pretty well ruled out unless he had surgical intervention, which has not been mentioned, as three years without symptoms pretty well separates it from the present illness.

The patient had cough, dyspnea, and weakness. There are several things that don't fit too precisely

* Completed residency in September, 1954. Now at Springfield, Missouri.
† Received M.D. degree in June, 1954.

but which, I think, should be mentioned. Among these are bronchiectasis. The patient did have a productive cough of three months' duration. I think that three months is a short time for bronchiectasis to become this severe. He had no hemoptysis. Other than during his hospital stay, we don't know how productive was this cough.

The patient apparently had close contact with tuberculosis. The weight loss, cough, and weakness are all good evidence that the man had an active pulmonary tuberculosis lesion. I see no evidence of it in the x-ray. That is, I see no evidence of a single lesion. I think that the man may have had a diffuse pulmonary tuberculosis.

Left heart failure is a possibility. There was no evidence of primary heart disease to produce such decompensation. He had a normal arm-to-tongue circulation time, which pretty well eliminates the probability of a left heart failure. The patient had neither physical nor historical evidence of a primary focus for a pulmonary infarction. The history fairly well rules it out. Although showing rapid progression, the last three months' course did not have the sudden onset you would expect to find in pulmonary infarction.

Mediastinal tumor could produce such a picture. The x-ray, as nearly as I can see, shows no mediastinal tumor. However, it is possible that he had one which was masked by the heart and other mediastinal structures. We have only an anterior-posterior view. The progression is rather rapid for most mediastinal tumors.

The x-ray picture is consistent with that of metastatic carcinoma—metastatic malignancy—but again we find no evidence of a primary lesion. The course is consistent with this, but I feel that the x-ray picture is not too consistent in that the lesions are not circumscribed.

Primary carcinoma of the lung could account for the patient's history, symptoms, and weight loss. But there is no appearance of a discrete lesion in the lung, which is the typical picture of primary carcinoma. I know of no carcinoma that I have seen or heard discussed that has started with such a diffuse picture. Again, having only AP views, it is possible that a primary carcinoma of the lung is not visible. But I don't feel that it would produce the x-ray picture we have. I think that if it were a primary carcinoma of the lung producing the symptoms of pneumonia, with the high white count, the changes in the lung would be more localized and more circumscribed. The patient is a male and in the right age group, which weights our thinking in favor of primary carcinoma of the lungs. Rapidity of progression does so, too.

However, the patient's history of tuberculosis, his rapid course, and the elevated white count, I believe, are sufficiently strong evidence of my final diagnosis of tuberculosis with a superimposed bacterial infection of unknown type.

Dr. Delp: Mr. Sandidge, do you think the fact that the findings were plucked out of the protocol may have influenced Mr. Coble in his final diagnosis?

Mr. Lewis Sandidge:* I'll say this much—after seeing the x-ray I changed my opinion—I think he probably changed his also.

Dr. Delp: What is your best diagnosis now?

Mr. Sandidge: My best diagnosis now is tuberculosis. Coble mentioned that there is no type of carcinoma that would fit that picture. I'm not sure, but I think that there is a carcinomatosis—a pulmonary carcinomatosis—that would produce a picture like this.

Dr. Martin FitzPatrick, assistant professor of medicine: The essential feature to consider in this patient is that we have a 66-year-old Negro male who had been in relatively good health all his life and in the last four months had been struck down with a severe pulmonary ailment that finally killed him. I think there are a few points that should be brought out carefully in his history.

One, of course, is the obvious one, that he had lived in a tuberculous environment. You will want to know if he lived in the same room, the same house, with the patients while they were having active pulmonary tuberculosis. The mere historical documentation that members of the family had the disease really does not mean that the patient was in intimate contact. This is a good suspicion, but I think you must go farther.

The other thing that I am uneasy about is a documentation of his work habits other than tending bar. From the time he left his family unit to go out into the world to work until he came back into the hospital, did he at any time work or be around any conditions that are the common cause of pneumoconiosis, such as silicon, asbestos or beryllium, bauxite, or diatomaceous earth? Any or all of these things can show a terminal picture like that presented today. The only way you can rule them out is by an adequate history.

Again, you want to know, in an elderly individual with progressive and fulminating disease, what his habits were in regard to the use of oil. Did he use oil nose drops? Did he use nose drops purchased at a drugstore over the counter without a prescription? Most of those are in an oily base. How often did he use them? Did he use oil to spray his throat for lubrication purposes? That is what happens with some singers. Did he use oil for catharsis? This was an

* Received M.D. degree in June, 1954.

individual who has absent dentition. He is described as having a poor gag reflex, and he aspirated before he arrived in the hospital.

I think you must be on your toes as to the possibility of whether he aspirated before he came in—specifically, whether he aspirated oily or fatty products.

I will briefly run through some of the disorders that could produce the terminal picture we see on the x-ray. I will consider for a moment some of the inflammatory ones. First of all, bronchiectasis. If this was bronchiectasis, I think it must have been diffuse. It is true that most of the infiltrations we see on the x-ray were in the lower half, but if you look at the films closely, you see a fine reticular pattern in the upper lung fields as well. Of course, it has been mentioned previously that the history in regard to sputum production, reputed pulmonary infections, possibly hemoptysis, going back to childhood, is lacking in this individual. I don't think we shall belabor that point further.

Now we come to tuberculosis, which after all is the mother of all chest diseases. I think there is the possibility of tuberculosis, judging from the history. He could have had phthisis: cavitary pulmonary disease with a rapid bronchogenic dissemination leading to multiple areas of tuberculous involvement in all his lungs. I think you would anticipate seeing a cavity on the casual x-ray. The sputum should have had pus in it. He should have been febrile. He should have been a lot sicker and toxemic and infected than this patient was when he was first seen at this hospital. So, in spite of his family history, on the limited information we have, I am ruling out tuberculosis. With adequate cultural studies we could always rule it out. For the purpose of argument, let's do that.

I think another thing we must consider are the suppurative pneumonias. A diffuse suppurative process that is generally superimposed on a chronic bronchitis or on a chronic bronchiectasis can lead to a terminal film picture similar to this. Now that again has a prolonged period of chronic pulmonary symptoms, of cough, of raising pus and purulent sputum, of repeated low grade infections, and of visits to the hospital. None of those were present in this patient. So I would be inclined to rule out chronic suppurative pneumonia in both his lungs. The same thing would hold true regarding acute pneumonia due to the common bacterial offenders.

Pneumoconiosis—I have belabored that point enough. I do not like to turn my back on that as a diagnosis from an x-ray in an individual without an adequate history. But let us assume for the purpose of argument that he had not had any known exposure to any of those agents.

Some of the fungus diseases could do this. I think it is well to remember that occasionally disseminated coccidioidomycosis, histoplasmosis, blastomycosis, or actinomycosis can mimic this in the x-ray picture to some extent. Such people are sicker. They present a more toxic state than was seen in this patient. Again, you can only rule them out with adequate cultural bacteriological studies.

The diseases of unknown etiology should be mentioned. I have seen a similar picture in scleroderma. It is not as fulminant as this but is rather a diffuse pulmonary fibrosis. It causes marked disturbance in alveolar respiratory gas exchange and little damage to ventilation. Such individuals have a profound respiratory deficit; they also show the classic skin findings of scleroderma as well as esophageal findings. We are told that the esophagus was normal in this patient, and so I think we can eliminate scleroderma as a cause of his alveolar difficulties.

Sarcoidosis, I think, would be unusual in this individual. He had no peripheral lymphadenopathy. He had no enlargement of bronchopulmonary or mediastinal nodes. His globulin was normal. He had no other systemic involvement, such as involvement of skin, salivary gland, or uveal system. That, I think, eliminates sarcoid.

That brings me back to lipoid pneumonia. I feel that we are missing this disease in many elderly people because we are not looking for it. We know that in two age groups, the very young and the very old, patients will aspirate oil and fat products and have a chemical pneumonitis set up in their lungs leading to severe fibrosis, incapacity, and sometimes death. Without that information, I would tend to rule out lipoid pneumonia. The few individuals I have seen with it have been sicker than this patient was. They have been febrile and have had pleural involvement. So let's eliminate lipoid pneumonia.

Another thing I think should be considered is aspiration pneumonia. We realize that practically all pneumonias are aspirational, but I refer specifically to the picture seen in elderly individuals with absent teeth and a poor gag reflex. The great watchdog of the tracheobronchial tree is eliminated, and these folks will unconsciously aspirate from time to time enough stuff within the tracheobronchial tree to leave a pulmonary fibrosis. The lesions of the esophagus are notorious for this. Over a period of years, the smooth or benign tumor, the leiomyosarcoma, can produce a severe form of pulmonary fibrosis simply from the repeated unconscious aspirations of the patient at night when he is asleep and his cough reflexes are minimized. In some of these folks the presenting picture can be that of pulmonary disease. With the normal esophagogram in this individual, I think we can rule that out.

The question of multiple pulmonary emboli, either

of blood elements or of tumor, is worth considering. In this individual there was no suggestion from either history or physical examination of an obvious focus supplying emboli to the internal blood vessels to his lung. The people I have seen with such a disorder have been a lot sicker, most of them febrile. Most of them occasionally have shown hemoptysis, severe chest pain, and a few other things lacking in this patient's picture.

Carcinoma must be considered. Some mention has been made of the fact that this would be an unusual site for a primary bronchogenic carcinoma.

Usually primary bronchogenic carcinoma shows symptoms of cough or bronchial obstruction. Metastases from carcinoma arising in another organ and brought to the lung, I think, can be ruled out because of the fact that it was not brought out in the history. There was no suggestion of it in the history or on the physical examination. The fact that this man had a normal gastrointestinal profile on barium studies would rule out lymphatic dissemination of carcinoma.

We're getting very far out on a limb. There isn't much left. One thing I think we can say about this patient—I think it's the same point that Dr. Herbert was getting at earlier—is the striking history, and this is what you must build your whole house on. If it's true, you've got a fine house. If it's not, it comes crashing down like one made of a deck of cards. But if you can be sure that, up until four months before this patient came in and died, he was relatively symptom-free as far as his lungs are concerned, then there aren't too many things that can do that.

First, I think, in this patient we must seriously consider what has been previously described as pulmonary adenomatosis: a diffuse multicentric tumor arising in practically all portions of the lungs. We don't know too much about this disease. It is thought to be similar to the infection jazziekte in sheep. It is thought by others to be possibly viral in origin. At any rate, these folks show a clinically profound dysfunction in their physiology, particularly in regard to respiratory gas exchange, without much defect obvious in their ventilatory function until later.

In the few cases I have seen, the sputum is extremely characteristic, with copious quantities of sputum that is white, viscid, and opaque, much like the lining on opal glass light fixtures. It has a high protein content, and when you take the glass jar and invert it you can put it back down without fear of spilling—it is that thick. Lacking that in this individual, and being told that his sputum is nondescript, nonspecific, the mucoid sputum that anyone raises, I must turn away from pulmonary adenomatosis.

That brings me to my last entity which I believe this fellow had, a rare disorder. I want to stress that point because when you are working in a hospital where there are a large number of pulmonary problems, you probably won't see more than one or two of these in several years. It is the picture I think this individual had, described by Hammond and Rich in 1944, which they called "acute diffuse interstitial fibrosis of the lung." It's a syndrome that they described as an acute form of pulmonary disease appearing in people who previously had been well. They are struck down without any warning, usually with a non-productive cough, later on raising sputum, with a severe derangement of their pulmonary physiology. They become hypoxic, cyanotic, and weak, and they lose weight. Many such patients earlier were afebrile and later on died of either a terminal bronchial pneumonia or from an acute cor pulmonale. I feel that fits this individual best.

I would reconstruct it by saying that here was a 66-year-old man with diffuse vascular disease. I think he probably had cerebral atherosclerosis. I would explain his previous symptoms on the basis of cerebral thrombi leaving some residuum. Superimposed on that framework, I think this individual acquired the disease described, acute diffuse interstitial fibrosis, and rapidly succumbed to that. I think terminally he was helped out by bronchopneumonia (the old man's friend), and also by congestive failure.

We are told that when this man came into the hospital he was afebrile and he was not in failure. Yet before he died he developed a picture of congestion. I think there is strong possibility that he might have developed acute cor pulmonale. And there is a possibility that he might have had myocardial debilitation and failure on a hypoxic basis.

Dr. Delp: Our guest is Dr. Stewart from the National Cancer Institute. Dr. Stewart has a keen interest in one specific area of this problem. (I must apologize to the senior for not letting him know that Dr. Stewart would participate. I was afraid that I might influence him.) Dr. Stewart?

PATHOLOGY REPORT

Dr. Harold L. Stewart, visiting pathologist:* It is pretty obvious that I'm not here to discuss pulmonary pneumoconiosis or anything like that.

This patient died of adenocarcinoma of the lung.

During the clinical course of the patient, Dr. Pollack examined the sputum for carcinoma cells, and she made a positive diagnosis of adenocarcinoma in this case.

The specimen which we will see has nodules throughout all lobes of the lung. These nodules are

* Chief, Laboratory of Pathology, National Cancer Institute, Bethesda, Maryland.

hard and circumscribed and there are many of them—they vary from 1 to 2½ cm. in diameter, most of them. Some of them have a peculiar hollowed-out central cavitation, and the inner aspect of that cavity is smooth as though it were lined by alveoli. There is in the gross note of the autopsy protocol the information that when these lesions were squeezed they exuded a mucoid material.



Figure 3. Gross photograph of cut section of lung, right lower lobe, containing several tumor nodules. Note the pattern of central cavitation with surrounding radiation of tumor.

Now there was no real primary tumor in this case, in the sense that most lung cancer arises in a bronchus, is often visible in the x-ray, and then there is secondary spread throughout the lung from that particular area. As you can see from the x-ray and, as a matter of fact, from this specimen, there was no good primary of that type where it could be demonstrated in the bronchus with the characteristics of a primary cancer of the lung.

However, in the upper portion of the lower lobe on the left side, there was the largest mass of tumor that was seen in this patient—an area about 1½ x 2 inches—but even that was not one continuous nodule. It consisted of a fairly large nodule and four or so smaller nodules. There was a good deal of retraction of surrounding lung tissue. The bronchi have been carefully explored in this specimen, particularly next to this particular nodule. We traced out the bronchi as far as we could, and once they ran into the region of the nodule they seemed to be compressed and the lumens obliterated. This was borne out also in the microscopic sections. In many areas the bronchi actually were compressed. One reason for this, as we can see from the microscopic pictures, is that there was extensive lymphatic invasion in the lung, bronchi, and around the blood vessels. You could see tumor cells within some of the larger bronchi and there were tumor cells within the blood vessels in this lung.

In other words, we are faced here with a problem. Is the tumor primary in this one nodule in the upper lobe of the left lung? Or is this one of those so-called multicentric primary tumors, so-called bronchiolar carcinoma related to pulmonary adenomatosis by some? That's really the problem as the pathologist is faced by it here (and I'd like to say something about it when I show the sections).

There was extensive lymphatic permeation subpleurally. There were a few lymph nodes around the bronchi—and I believe I saw one around the aorta—containing cancer. I think there were two or three in the left cervical region that showed cancer. But as a matter of fact the extent of involvement of the lymph nodes by cancer was relatively minimal. The peripheral sinuses were infiltrated, and there was some extension into the surrounding fat, but there was no large amount of replacement of nodes by cancer tissue. I thought in the left cervical node there was involvement of a blood vessel, but there was a question of whether that might be one of the larger lymphatic vessels. But really the lymph nodes did not show much involvement at all.

Elsewhere in the body, either in the lower thoracic or the upper lumbar vertebra, there was a small solitary metastasis of carcinoma. That was the only tumor that was found outside of the lung and cervical lymph nodes—this one deposit in one of the vertebra. There was, of course, a careful search of other organs that might yield metastases of this type to the lung, the pancreas, the bile duct, the entire gastrointestinal tract, and they showed none.

Another striking feature about the lung in this case was an extensive and widespread thrombosis of the blood vessels. Those thrombi were of varying ages. Some of them were old; some of them were fresh. There were red thrombi and mottled thrombi; there were thrombi in varying degrees of organization and recanalization. So I felt certain that some of these thrombi in the vessels of this lung had been there for a considerable time. We did find a source for emboli in the prostate, but the thrombi in the prostatic veins appeared to be fresh. Certainly the thrombosis of the vessels in this case was extensive, and that process had been going on for some time.

Despite the pulmonary vascular lesions, there was no cor pulmonale in this case. The right ventricle was dilated, but there was no right-sided hypertrophy.

There was some hypertrophy of the heart—I believe the heart weighed 450 grams. The coronary vessels showed moderate arteriosclerosis and some narrowing in the size of the lumen in some cross sections. But there was no evidence of any gross infarction.

In addition the patient showed arteriosclerosis of the aorta and a little arteriosclerotic atrophy of the kidneys. There was a hyperplasia of the prostate, and there were thrombi in the prostatic vessels. I noticed one small infarct in the prostate about one cm. in diameter.

The gastrointestinal tract and the pancreas showed nothing of note.

The liver was interesting. There was some fatty central congestion and infiltration with hepatic cell necrosis. Of course this man was in cardiac failure. Then there were a few small nodules that looked a little like adenomas in the liver, and yet microscopically there were bile ducts and portal areas within these—a curious sort of lesion which may have represented adenomas or perhaps merely regenerating nodules.

In the brain there was severe arteriosclerosis of the vessels. There was an extensive area of old softening of the brain in the right parietal region of the cortex. There was a vessel leading to that which was completely occluded. In the internal capsule on the left side there was an area of degeneration and atrophy—an old scarred area. I presume that the patient's residual paralysis on the right side was due to the lesion in the internal capsule.

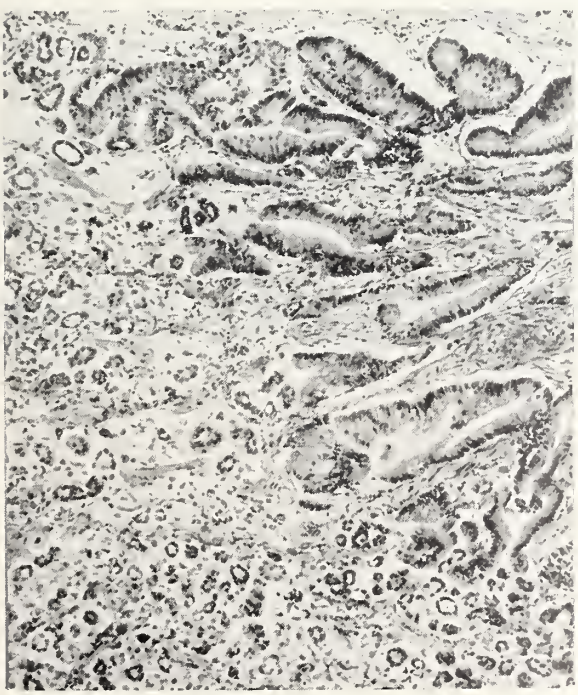


Figure 4. Photomicrograph of section through typical tumor nodule in lung, showing central tubular structure (on the right in photograph) lined by tall columnar epithelium resembling bronchial mucosa with rosettes around the periphery (on the left in photograph).

This gives you the general diagnosis and important lesions.

On microscopic examination of the lung, some localized areas of the tumor were cavitated and others were fibrosed and burned out.

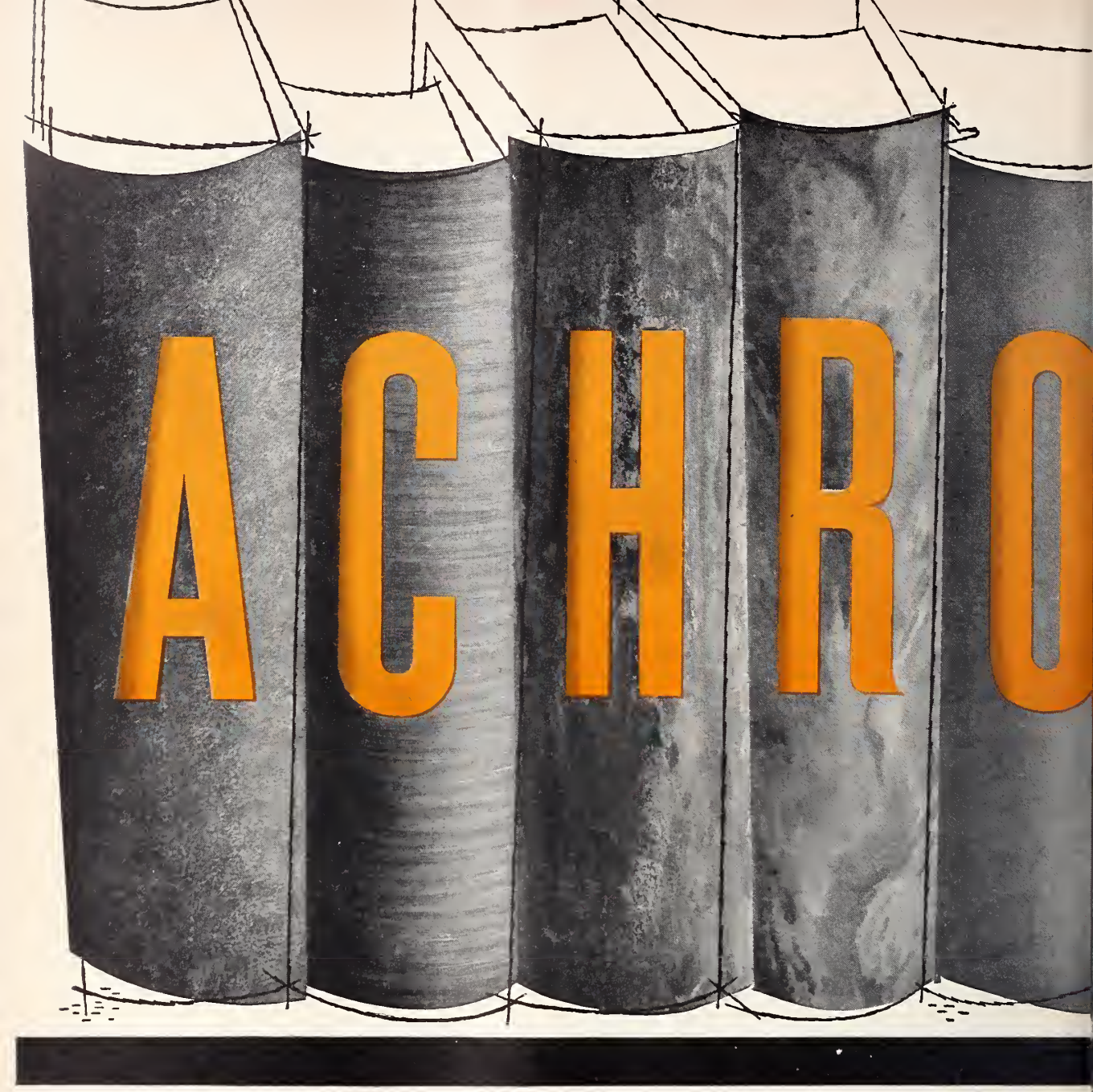
The Periodic Acid Schiff reaction was positive, demonstrating the mucus production in this tumor. Some of the cavities actually contained an enormous amount of mucus. Now whether it was due entirely to production of mucus by the tumor cells or whether in part due to obstruction of the bronchus and accumulation of mucus, I don't know. Perhaps both were operative.

There's a question about the origin of this tumor. As you know, a so-called bronchiolar carcinoma (at least, it's classified as that) is a lesion that's often multiple throughout all lobes of both lungs. It's related on one hand to jazziekte, so-called pulmonary adenomatosis, and on the other hand to primary adenocarcinoma of a main bronchus. The so-called bronchiolar carcinomas have been given separate designation because a rather small per cent, probably 3 or 4 per cent, are believed to be multicentric in origin.

There is a great deal of dispute about that. Fred Stewart, for example, at Memorial Hospital, in analyzing their cases, showed that the postoperative survival of these cases is better than in the localized type of adenocarcinoma, which makes him believe that these are not multicentric but that they are metastases from one area. If they were multicentric, there would be no hope—they would arise all through all lobes of both lungs. And even though you took one lung out or one lobe out, the patient would still develop tumors in other areas.

There was nothing in this lung that I have seen that indicated to me the development of cancer—that is, the pre-cancerous state, a terminal bronchus undergoing a hyperplasia that leads to cancer. I saw lots of cancer in the alveoli; I saw it in the perivascular and peribronchial lymphatics. But there was no place where I saw, at the junction of a terminal bronchus and alveolus, changes that I would interpret as a beginning of cancer. Certainly there is a possibility that this large lesion in the left lung was the primary site, that the spread throughout the lung was by the lymphatics, and that all the rest of this represented secondary deposit.

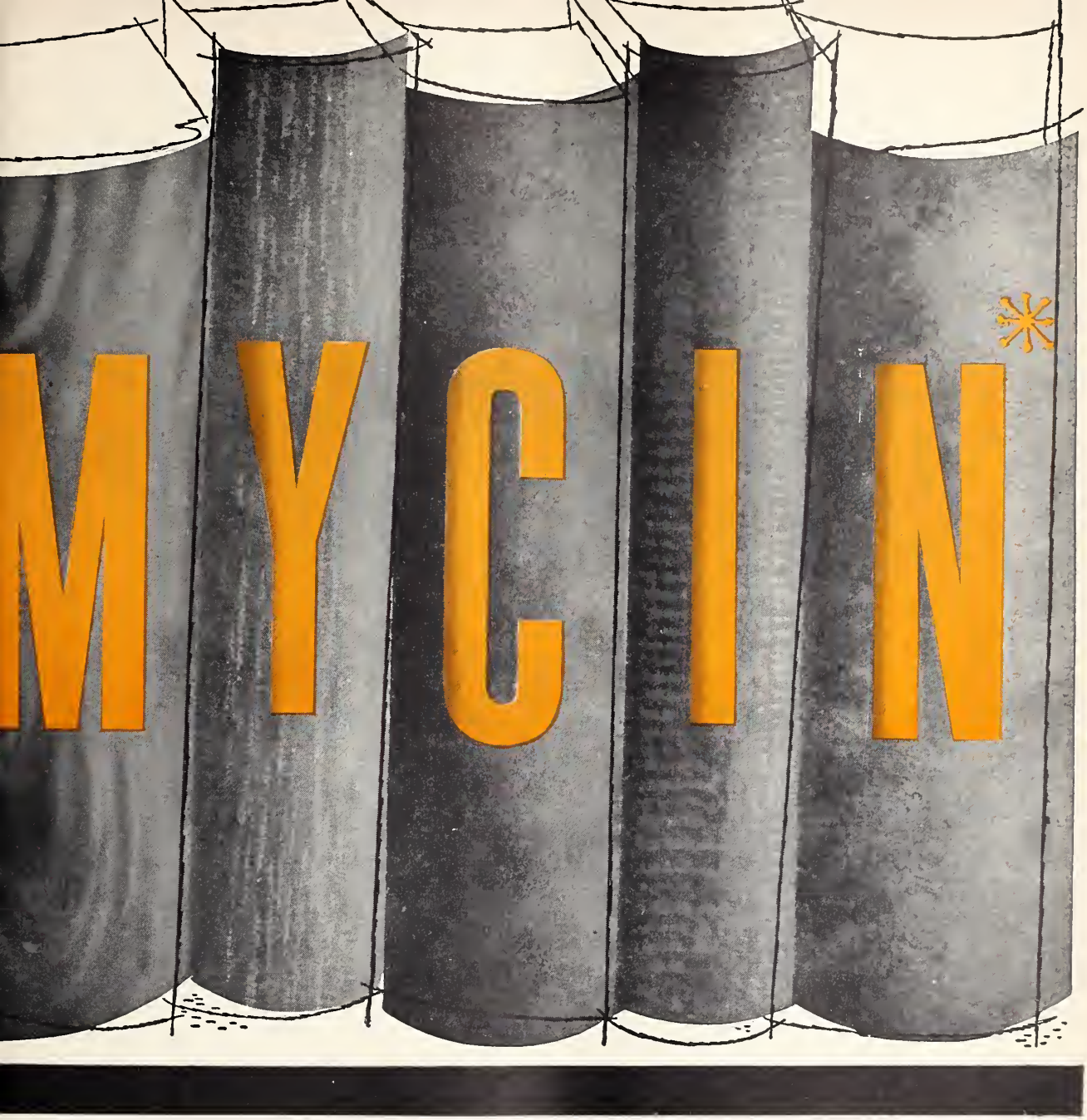
There is one other possibility, too, and that is dissemination by the air passages. Ordinarily that just does not occur—we know that. For example, in cancer of the larynx, in cancer of the tongue, in most cancer of the lung, we know that dissemination by the air passages simply does not occur. But I must



Medical history is being written today

Lederle

® REG. U.S. PAT. OFF.



Hydrochloride
Tetracycline HCl *Lederle*

The introduction and rapid widespread adoption of ACHROMYCIN has opened a new chapter in the history of broad-spectrum antibiotics.

ACHROMYCIN fulfills the requirements of the ideal antibiotic in virtually every respect . . . wide-range antimicrobial activity, *in vivo* stability, tissue penetration, minimal toxicity.

ACHROMYCIN is truly a broad-spectrum weapon, effective against Gram-positive and Gram-negative bacteria, as well as certain mixed infections.

ACHROMYCIN is more stable and produces fewer side effects than certain other broad-spectrum antibiotics.

ACHROMYCIN provides prompt diffusion in body tissues and fluids.

ACHROMYCIN is destined to play a major role among the great therapeutic agents.

LEDERLE LABORATORIES DIVISION AMERICAN Cyanamid COMPANY PEARL RIVER, NEW YORK

admit in this case there were suggestions of that in the slides. The cancer cells were lying inside the bronchi, even the major bronchi. You could see a small alveolus and you could see cancer cells in there, often against the wall of the alveolus. This case certainly suggested to me the possibility that the primary focus was in the left upper lobe and all the rest of this represented metastasis via the lymphatics and possibly the air passages.

Dr. Delp: Dr. Firminger, do you have any comments?

Dr. H. I. Firminger, pathology and oncology: I will just register the opposite opinion that this probably was a tumor arising multicentrically. I have two reasons for my opinion. One is that I am loathe to accept air spread of tumors, though I must admit that maybe I am biased by the fact that most of them don't do it. And I cannot explain to my own satisfaction so wide a spread into both lungs of this tumor by lymphatic channels without more involvement of hilar lymph nodes—replacement of hilar nodes. It is conceivable, but somehow it doesn't quite fit.

And finally, a somewhat more subtle thing, each of these individual nodules appeared to me as though it must have arisen there, though I can't show you the bronchus or the terminal bronchiole from which it came. The reason I say that is because of the picture Dr. Stewart so well illustrated of the central cavitation which was evident in these lungs, with degeneration around it and then a radiating pattern of what looked like bronchiolar epithelium which then differentiated into alveolar duct-like epithelium. This two-cell pattern, which was consistent throughout, of central bronchiolar type and then, if I may use the term, alveolar duct type of epithelium beyond it in practically all the nodules seems so consistent and it seems to have arisen in all these places at once.

I would prefer to think of this as a multicentric tumor, but it is clearly a moot point.

The picture does fit pretty well with pulmonary adenomatosis, except for the widely disseminated lymphatic spread. It is not particularly pointed out in the literature, but in my own experience one does not usually find this picture at autopsy without at least metastasis by the lymphatics to the hilar lymph nodes, which was present here.

Dr. Stewart: There are a couple of things to be mentioned about this. One is that there really was an abrupt history in this case. This man came in and died, and as far as we know he was in pretty good shape before that. If you think of multicentric carcinoma, it would be amazing if you'd get carcinoma in at least 150 sites as in this case, all arising at these different sites within a short space of time, say a month. I mean it would be extremely unlikely, because this patient's symptoms were due to the carcinoma that he had all over his lungs, not to the one lesion in the one lobe. And it's much more logical to me that he slowly developed a carcinoma of the left upper lobe and that he suddenly had a terrific spread of that to the rest of the lung.

We know he had metastases. He had metastasis to the mediastinum, outside the mediastinum to the cervical lymph node, and to the vertebra. We found tumor cells in the blood vessels. We found them in the lymphatics. Furthermore, nobody can show me in this specimen exactly where this tumor arose. As a matter of fact, before I would accept it as multiple primary, I would have to see some sites in which this tumor was arising. If this tumor had arisen in multiple sites, certainly there must be other sites where it was just arising when the patient died. And they are not present in that specimen.

Dr. Delp: Dr. Stewart, we are grateful to you, and I think I need say nothing more about this case.

ANATOMICAL DIAGNOSIS

Primary

Primary adenocarcinoma of lung, involving all lobes of both lungs with metastases to anterior cervical, periaortic hilar, tracheobronchial and mediastinal lymph nodes and vertebrae (history of chronic cough and marked dyspnea and orthopnea for two months before death).

Organizing thrombi (? emboli) of many branches of small pulmonary arteries, bilateral.

Arterionephrosclerosis, slight.

Hypertrophy of the heart, 450 grams, and dilatation of the right ventricle, moderate (history of hypertension for at least 12 months before death).

Arteriosclerosis of aorta, cerebral and coronary arteries, advanced.

Organized and recanalized thrombus of right middle cerebral artery.

Encephalomalacia of the parietal lobe of the right cerebral hemisphere and of the left internal capsule (history of cerebral thrombotic episodes 12 and 8 months before death, with residual weakness of right arm and leg).

Multiple petechial hemorrhages in mucosa of stomach, small and large intestines and ecchymoses in mucosa of small intestine.

Acute passive congestion of liver, spleen, and kidneys, moderate.

Edema of the lower extremities, advanced (history of multiple venipunctures).

Focal fatty metamorphosis of the liver.

Accessory

Focal fibrosis of heart.

Chronic bronchitis, slight.

Focal atelectasis and emphysema of all lobes of both lungs.

Congenital absence of middle lobe of right lung.

Multiple small adenomas of the liver.

Small calcified nodule beneath the capsule of the left lobe of the liver.

Nodular hyperplasia of the prostate, advanced, with small medial bar formation.

Focal area of infarction of prostate.

Multiple thrombi of veins in prostate.

Hypertrophy and trabeculation of wall of urinary bladder.

Cancer Society Meeting

The annual meeting of the Kansas Division, American Cancer Society was held at the Hotel Lassen, Wichita, October 10 and 11. Speaker at the banquet session was Mr. Edward F. Reid, Andalusia, Alabama, a cured cancer patient who devotes a large part of his time to work for the cancer organization.

At the business session Dr. Karl E. Voldeng, Wellington, was re-elected president. Mrs. Tom Stewart, Wellington, was named vice-president; Mr. Arthur H. Saville, Topeka, chairman of the executive committee; Mrs. Karl Leidig, Lenora, secretary, and Mr. Robert A. McClure, Topeka, treasurer.

Members-at-large of the executive committee are Dr. Howard E. Snyder, Winfield; Mr. Kean Tilford, Wichita; Dr. Robert H. Riedel, Topeka; Dr. Robert E. Stowell, Kansas City, and Dr. Orville R. Clark, Topeka.

Kansas Council for Children and Youth

The 12th annual meeting of the Kansas Council for Children and Youth was held at the University of Kansas Medical Center, Kansas City, October 14 and 15.

The address of welcome was given by Dr. W. Clarke Wescoe, dean of the school, after which Dr. Franklin D. Murphy, chancellor, discussed "The Institute for Exceptional Children." The following scientific program was presented: "Prevention of Poliomyelitis," Dr. Herbert A. Wenner; "Prevention of Rheumatic Heart Disease," Dr. A. M. Diehl; "Community Services of Child Study Unit," Dr. Harry G. Gianakon. Dr. Paul C. Laybourne and Dr. Edward D. Greenwood participated in a panel discussion on "Prevention of Juvenile Delinquency."

Subjects Announced for "Medic" Series

The new television program "Medic," sponsored by the Dow Chemical Company, will include the following subjects in the future: bubonic plague, November 22; hearing, November 29; unwed mother, December 6; traffic accidents, December 20.

DEATH NOTICES

Oliver David Walker, M.D.

Dr. O. D. Walker, 93, who served as president of The Kansas Medical Society in 1916, died at his home in Salina, October 6. He had retired from active practice about 15 years ago.

Dr. Walker was graduated from the College of Physicians and Surgeons, Keokuk, Iowa, in 1886, and he began his career as resident physician at the Haskell Institute, Lawrence. He then returned to Keokuk and remained there until 1905, teaching physiology, histology, and nervous diseases and serving as secretary of the faculty. He moved to Salina in 1905 and continued to practice there until his retirement.

Throughout his professional life Dr. Walker performed many services for medicine. His election to the presidency of the Society followed several terms as a member of the Council. He became a member of the Kansas State Board of Health in 1911 and later served as its president.

He was also an active participant in civic affairs, having spent a great many years as a member of the Board of Education in Salina and 10 years as its president. He was a charter member of the Salina Rotary Club.

James Carey Robb, M.D.

Dr. J. C. Robb, 75, an active member of the Ford County Medical Society, died at the hospital in his home city, Fowler, on October 6 after having suffered a heart attack that morning. A graduate of the Keokuk, Iowa, Medical College in 1901, Dr. Robb received his Kansas license in 1905. Since 1908 he had practiced in Fowler, and in 1950 he was honored at a community celebration recognizing his "valued and faithful service to his community."

Dr. Robb found young people of special interest, and he served as coach for one of Fowler's early football teams. He had also served on the school board in the community and was active in several fraternal organizations.

ACTIVITIES OF MEMBERS

Dr. A. M. Cherner, Hays, was principal speaker at a recent meeting of representatives of the American Cancer Society in that area.

Dr. Frank F. Merker, former chief of continued treatment at Winter VA Hospital, Topeka, has been appointed acting chief of professional services there.

Dr. Oscar W. Davidson and Dr. Lee H. Leger, Kansas City, have been appointed to serve as members of the board of directors of the Student Union and Dormitory Corporation of the University of Kansas School of Medicine.

Dr. Mary T. D. Glassen, Phillipsburg, has been chosen as chairman of the Kansas Council for Children and Youth.

At a recent meeting of the American Academy of Ophthalmology and Otolaryngology held in New York City, Dr. LaVerne B. Spake, Kansas City, was honored with the presentation of the organization's award of merit, a gold key.

Dr. Thomas L. Foster, Halstead, discussed "Understanding Each Other in the Home" at a recent Republic County Family Life Institute held at Belleville. He also spoke on "Mental Health Is Salina's Business" at a meeting of the Saline County Association for Mental Health.

Dr. Cora S. Crews, who has been practicing in Hiawatha, has moved to Iola and is now practicing there in association with Dr. Russell Nevitt.

Dr. John M. Porter, Concordia, addressed the Rotary Club in Kansas City on October 5. He told of changes that have taken place in methods of caring for the sick during the past 50 years.

Dr. Jere O. Osborne, who moved to Ness City in July, has announced the opening of an office there.

Dr. Stanley R. Friesen, associate professor of surgery at the University of Kansas School of Medicine, was recently elected to membership in the American Surgical Association and to associate membership in the American Association for Thoracic Surgery.

Dr. Carroll D. Behrhorst, Winfield, and Dr. Roscoe F. Morton, Arkansas City, were recently named part-time health officers for the northern and southern divisions of Cowley County.

Dr. Ernest P. Carreau was recently discharged from duty with the Navy and has returned to his practice in Wichita.

Dr. S. Albert Scimeca, who formerly practiced in Caney and in Wichita, has announced plans to open an office in Bucklin.

Dr. Roscoe F. Morton, Arkansas City, addressed the Lions Club there recently and compared methods of drug manufacture and medical care in Europe with methods in this country.

Dr. Alfred M. Tocker, Wichita, was speaker at a recent meeting of the Sedgwick County Medical Assistants' Society. His subject was "New Techniques in Cardiovascular Surgery."

Dr. George Lockett, formerly of Glasco, is now practicing in Moline.

Dr. J. Dean Gough, Chanute, addressed a recent meeting of nurses at Neosho Memorial Hospital on the subject of diabetic conditions.

Awards for service to the Kansas Tuberculosis and Health Association were presented to five physicians at the annual meeting of the organization in Kansas City last month. Those so honored were Dr. F. A. Trump, Ottawa; Dr. Dellett E. Bronson, Olathe; Dr. Clinton R. Lytle, McPherson; Dr. Clay E. Coburn, Kansas City, and Dr. Ralph I. Canuteson, Lawrence.

Dr. Hansel Benvenuti, medical director of the Sedgwick County Hospital, Wichita, during the

past three years, has announced plans to leave on December 1 to take charge of medical service at the Metropolitan Hospital, near Los Angeles.

Dr. Ballard Hayworth, who practiced at the Topeka Medical Center before he entered military service, has announced plans to practice in Redwood City, California, on his release from service.

Dr. Henry O. Hardesty, Jennings, who had practiced 54 years before he retired last summer, was the subject of a feature story in the *Norton Daily Telegram* on October 15 in connection with the observance of his 85th birthday.

Dr. Edmer Beebe, Olathe, announces that Dr. J. Robert Twinem, formerly of Garden City, is now associated with him in practice.

Dr. Franklin D. Murphy, chancellor of the University of Kansas, was featured speaker at the sixth annual Kansas Conference on Family Life held at Wichita, October 23.

Dr. A. E. Hiebert, Wichita, was speaker at a meeting of the Sedgwick County unit of the American Cancer Society on September 30.

Dr. Eugene Walsh, Onaga, has been appointed health officer for Pottawatomie County.

Dr. Donald L. Rose, Kansas City, has been elected third vice-president of the American Congress of Physical Medicine and Rehabilitation. Dr. Rose also serves as a member of the Board of Governors of the Congress.

Dr. Jack R. Cooper, Kansas City, addressed a meeting of nurses of District 2 at Bethany Hospital last month. His subject was neurosurgery.

A.M.A. Stand on Reinsurance

A résumé of the stand of the American Medical Association with regard to the government's reinsurance proposal was prepared last month by Dr. George F. Lull, secretary and general manager of the association. While in complete agreement with the objective, "to encourage and stimulate private initiative in making good and comprehensive health services generally accessible on reasonable terms," the A.M.A. opposes reinsurance for the following reasons:

1. The mechanism suggested would not accomplish the stated purposes of the bills.
2. The phenomenal progress of the health insurance industry makes federal intervention not only unnecessary but a dangerous intrusion into a successful area of private enterprise.
3. "Reinsurance" would not make health insurance more attractive to persons who can afford to pay premiums and have not done so. It would not make health insurance available to the indigent unless the government provides a subsidy for the purpose of selling insurance at less than the cost of servicing the contract.
4. The program, without subsidy, would not make health insurance available to any additional groups or geographic areas that voluntary insurers cannot reach.
5. Most insurance authorities agree that the extent of health insurance liability is such that a federal reinsurance program is absolutely unnecessary.
6. The bills would give the secretary of the Department of Health, Education and Welfare an extensive but unjustified regulatory control over the nation's health insurance industry.

Psychiatric Association Meets

The annual meeting of the Mid-Continent Psychiatric Association, branch society of the American Psychiatric Association for the states of Arkansas, Kansas, Missouri, and Oklahoma, was held in Little Rock, September 18 and 19.

Dr. Paul Hines, Kansas City, Missouri, was elected president. Other officers are: Dr. Harold W. Sterling, North Little Rock, vice-president and president-elect; Dr. Frank F. Merker, Topeka, secretary-treasurer, and Dr. William Rottersman, Topeka, councilor for Kansas. Dr. Rottersman took part in the program, presenting a paper, "New Hope for State Mental Health Program."

Nearly 400,000 nurses, an increase of 16,000 since 1950, are now working in the United States, according to figures released recently by the Public Health Service. Although 389,600 of the total are now in active practice of their profession, the demand for nursing services is so great that the present recruitment goal for the nation is 55,000 student nurses a year.

COUNTY SOCIETIES

Dr. V. C. Wiksten, Topeka, was guest speaker at a recent meeting of the Montgomery County Medical Society. He discussed allergies.

Members of the Cancer Committee of the Sedgwick County Society presented the program at the October meeting of the organization. Dr. F. L. Menehan served as moderator. The following speakers took part: Dr. C. T. Hinshaw, "Respiratory Tumor"; Dr. T. C. Hurst, "Local Incidence Statistics"; Dr. J. G. Lockhart, "Neuromas and Neurofibroma"; Dr. W. F. McGuire, "Kidney Tumors," and Dr. V. L. Scott, "Leukemia."

The society's 28th annual tourney was held this fall at the Rolling Hills Country Club. Dr. H. Lee Barry and Dr. Robert P. Norris received golf prizes. A tournament dinner followed the sports events.

Dr. Alfred P. Bay, superintendent of the Topeka State Hospital, presented a group of speakers from the hospital at the October 4 meeting of the Shawnee County Medical Society, Topeka. Ten-minute presentations were given as follows: "Changes in the Topeka State Hospital Since 1948," Dr. William Simpson; "Admission Procedures," Dr. George W. Jackson; "Basis of Psychiatric Treatment," Dr. J. G. Graybill; "Workup of the Psychiatric Patient," Dr. William Tarnower; "Problems in Psychological Medicine," Dr. Aron Arnow; "Child Psychiatry," Dr. Joseph Noshpitz; "Out Patient Treatment—Advantages," Dr. Howard V. Williams, and "Training Programs," Dr. Paul E. Feldman.

The Shawnee County Medical Society was host to the Golden Belt Medical Society at a meeting held at the Jayhawk Hotel, Topeka, October 14. Dr. Richard C. Tozer, Topeka, presented a paper, "Care of Head Injuries," and Dr. William L. Valk, Kansas City, spoke on "The Clinical Evaluation of Renal Function."

A meeting of the Montgomery County Society was held on October 20 at the Coffeyville Country Club. Speaker of the evening was Dr. William G. Norman, who has been in practice for 56 years. Dr. Norman was named by the group as its "Doctor of the Year."

A meeting of the Wyandotte County Society was held at the Town House Hotel, Kansas City, October 19. Dr. Frank F. Allbritten, Jr., chairman of the department of surgery, University of Kansas Medical center, presented a paper on "Carcinoma of the Lung." The organization in Wyandotte County now numbers 209, 18 new members having been accepted during the first nine months of the year.

Members of the Marion County Society met at the Kingfisher Inn, Marion, October 5. Dr. Robert D. Wood, who recently opened an office in Peabody, spoke on "Pre-Anesthetic Medication."

A meeting of the Labette County Society was held at the Parsons Country Club on October 13. Speaker of the evening was Dr. Stanley S. Peterson, Springfield, Missouri, who discussed the Red Cross blood program.

Clinics on Television

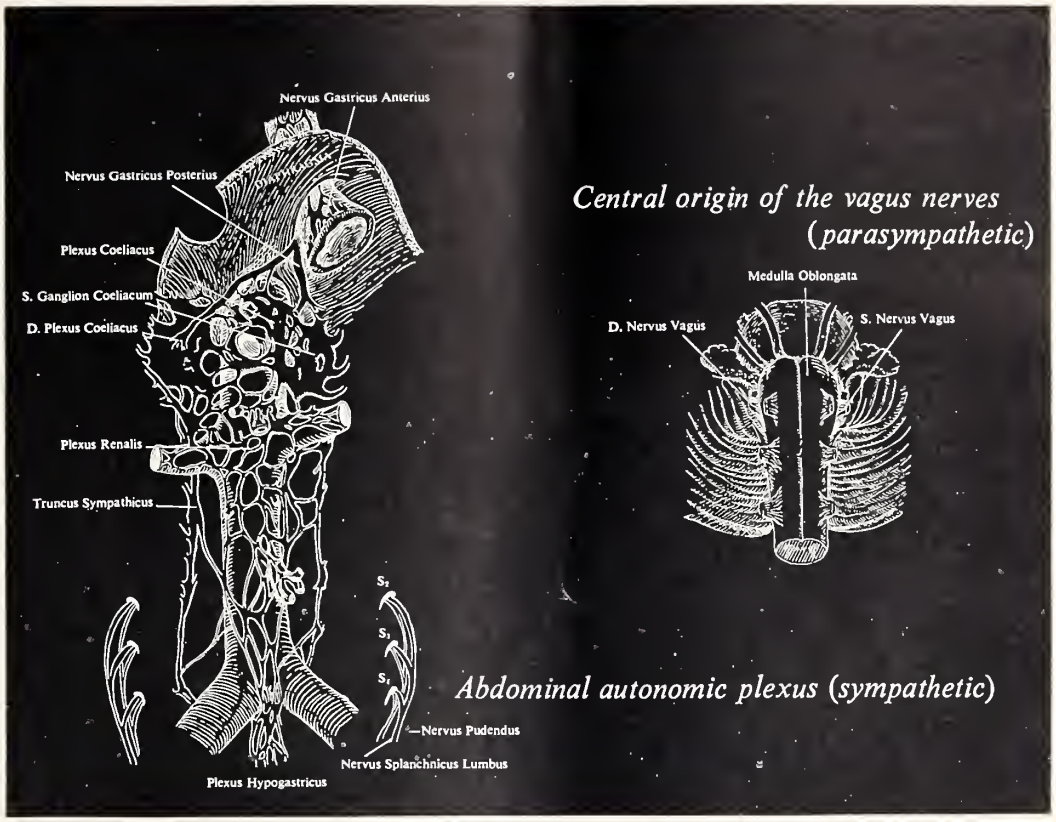
An experiment in the use of open-channel television for postgraduate medical education was termed successful when tried recently by the Division of Graduate and Postgraduate Medical Education of the University of Utah. With facilities and time made available by a Salt Lake City station, the university telecast "clinics" once weekly for four weeks.

The series covered x-ray diagnosis of calcifications in the abdomen, gastrointestinal hemorrhage in the elderly patient, the approach to congenital heart disease, and a diagnostic clinic. Programs were presented from 7:00 to 8:00 a.m.

A survey made later indicated that 48.9 per cent of the potential doctor audience viewed the telecasts. Seventy-four per cent of the viewers reported that they had learned new facts, 61 per cent felt that their "previous knowledge had been revitalized," and 53 per cent acknowledged renewed interest in a specific field.

The Kansas State Board of Health reported an increase in the number of cases of encephalitis in Kansas during the month of September. One city, Colby, reported eight cases, one of which was fatal. Other cases were reported from the following counties: Hamilton, Marshall, Clay, Butler, Jefferson, Bourbon, and Trego.

Blue Shield plans have passed the 30 million mark in enrollment nationally.



Control of Gastric Motility and Spasticity in Peptic Ulcer with Banthine®

"The need[1] for suppressing gastric motility and spastic states is . . . fundamental in peptic ulcer therapy. Since the cholinergic nerves are motor and secretory to the stomach and motor to the intestines, agents capable of blocking cholinergic nerve stimulation are frequently used to lessen motor activity and hypermotility."

Banthine[2] "has dual effectiveness; it inhibits acetylcholine liberated at the postganglionic parasympathetic nerve endings and it blocks acetylcholine transmission through autonomic ganglia."

It has been shown[1] to diminish gastric motility and secretion significantly as well as intestinal and colonic motility.

The usual schedule of administration in peptic ulcer is 50 to 100 mg. every six hours, day and night, with subsequent adjustment to the patient's needs and tolerance. After the ulcer is healed, maintenance therapy, approximately half of the therapeutic dosage, should be continued for reasonable assurance of nonrecurrence.

Banthine® (brand of methantheline bromide) is supplied in: Banthine ampuls, 50 mg.—Banthine tablets, 50 mg.

It is accepted by the Council on Pharmacy and Chemistry of the American Medical Association. Searle Research in the Service of Medicine.

---

1. Zupko, A. G.: Pharmacology and the General Practitioner, GP 7:55 (March) 1953.

2. McHardy, G. G., and Others: Clinical Evaluation of Methantheline (Banthine) Bromide in Gastroenterology, J.A.M.A. *147*:1620 (Dec. 22) 1951.

THE KANSAS PRESS LOOKS AT MEDICINE

Editor's Note. In this section the JOURNAL *reproduces editorials relating to medicine which have appeared in the lay press. An effort is made to include both favorable and unfavorable comments, and the Editorial Board in no instance assumes responsibility for the opinions expressed.*

The Coroner Question

Some of the brothers are giving out words about county coroners. Somebody brings up the proposition that the coroner system in Kansas should be overhauled. Specifically, the matter seems to have been raised because in many counties undertakers seek the office of coroner and frequently they are successful. A former legislator, a top pathologist in Kansas, thinks that Kansas should change its coroner system so as to make it necessary for a corner to be a physician. A doctor says that it is easy to get away with murder in Kansas. A murder may be committed and a coroner who is not a doctor will render an official decision that is not accurate.

A law to create county examiners, such persons to be appointed from a list of qualified persons by the county commissioners, is suggested. It is not the first time the proposal has been up. Two legislatures have defeated such a proposal. The defeats have been attributed to politics.

It is pointed out that the duties of a coroner have quite a vital effect on many matters including insurance. Permitting the job to go to someone who can get the votes to be elected without the qualifications creates a bad situation.

The coroner, it will be recalled, is the second most important officer in the county—at certain times. The most important is the sheriff. He is the chief executive officer of the county. If the sheriff cannot act or if he must be arrested, the coroner is the officer who takes over for the disqualified sheriff. That is of far less importance, however, than the responsibilities of the coroner in his ordinary run of events in the county. The proposal which is being made surely will have some merits. It is pointed out that it is possible for someone to be murdered by poison in Kansas without the public being advised. The coroner who is not a doctor will not know the difference between a fatal heart attack and poison. An autopsy would be required to get at the truth. With a physician as a coroner he can proceed with an autopsy. If he is not a doctor he cannot, obviously.

The office is one where politics or political attainments cannot substitute for professional knowledge and qualifications.

The next legislature could well afford to deal with this problem of the coroner system on its merits. —*Pittsburg Headlight, September 22, 1954.*

Aid for the Indigent

The American Medical Association has long been a whipping boy of those who want socialized medicine, or government-bossed medicine, or some variant thereof. The idea has been disseminated that this organization, which represents the vast majority of the country's physicians, is opposed to better medical care, and isn't at all concerned with the problems of indigent people.

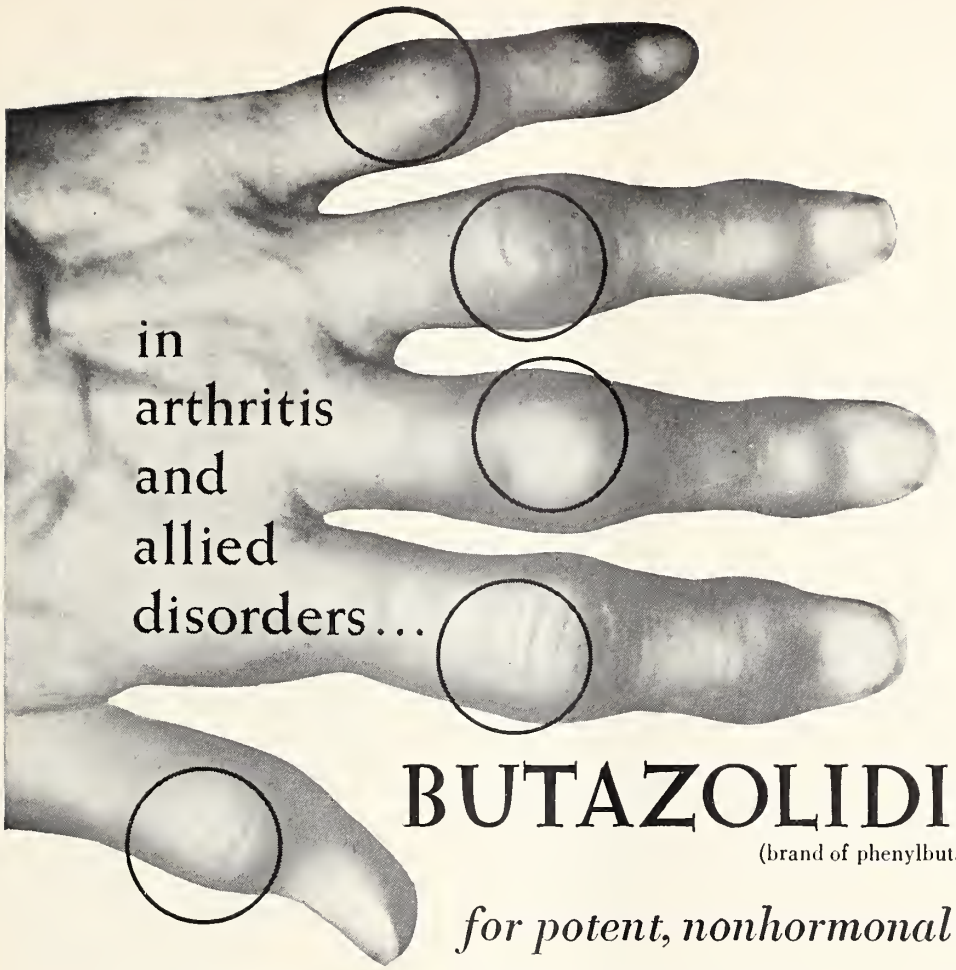
Nothing could be farther from the truth. The AMA has been directly and indirectly responsible for some of the great medical advances of all times. It has thrown its weight solidly behind the voluntary health insurance movement, and has been a positive factor in that movement's extraordinary growth.

What the AMA does oppose are schemes which would inevitably reduce the standards of medical care and put the bureaucrat in the driver's seat. A spokesman put the case accurately when he said: ". . . it is essential to consider two groups of individuals: (1) those who are able to pay the normal costs of medical care, and (2) those who are indigent. Most of the American people fall within the first group. . . . The other group—the indigent—poses distinctly different problems. . . . If the medical care problems of this group cannot be solved by the individuals or their families, the responsibility should be assumed by the local and state government."

Every community's problem is different, and can be solved locally. And aid for the indigent is a basic local responsibility—not a federal function.—*Industrial News Review, reprinted in Topeka Capital, September 18, 1954.*

A record graduation of 6,861 physicians during the past year by our nation's medical schools has boosted the ratio to an all-time high of one physician for every 730 persons in the United States. This ratio will be lowered even more in the next few years as the number of medical graduates is expected to rise.

Today's physician population has now reached approximately 220,100. Enrollment of 28,227 is the largest number of medical students in history in this country. The freshman class enrollment of 7,449 is also a record.



BUTAZOLIDIN®

(brand of phenylbutazone)

for potent, nonhormonal therapy

The anti-arthritic potency of BUTAZOLIDIN is well substantiated by recent clinical reports. In peripheral rheumatoid arthritis, for example, BUTAZOLIDIN produced "major improvement" in 42.9 per cent of the patients studied; in rheumatoid spondylitis "major improvement" in 80 per cent; and in gout 90.9 per cent demonstrated "marked improvement" or "complete remission of symptoms and signs within 48 hours."*

BUTAZOLIDIN being a potent agent, the physician should carefully select candidates for treatment and promptly adjust dosage to the minimal individual requirement. Patients should be regularly examined during treatment, and the drug discontinued should side reactions develop.

Detailed literature on request.

*MacKnight, J. C.; Irby, R., and Toone, E. C., Jr.: Geriatrics 9:111 (Mar.) 1954.

BUTAZOLIDIN® (brand of phenylbutazone): Red coated tablets of 100 mg.

GEIGY PHARMACEUTICALS
Division of Geigy Chemical Corporation
220 Church Street, New York 13, N. Y.
In Canada: Geigy Pharmaceuticals, Montreal 423

Some Basic Concepts of the Acute Radiation Syndrome

Eugene T. van der Smissen, M.D.
Baltimore, Maryland

Christmas eve, 1895, can be said to have ushered in an era that has revolutionized both medical and physical sciences. It was on this date that Professor Wilhelm Konrad Röntgen (Roentgen) announced his discovery of x-rays before the German Physical Society. Prior to this time men had experimented with the Crooke's tube, but Roentgen was the first to discover the "magic ray." From this beginning many divergent paths lead into the various sciences of physics and medicine.

The discovery of Becquerel rays in 1896 and the isolation of radium in 1898 were further steps along these pathways. Following the discovery of radium in 1898, physicists all over the world began to seek explanations of the cause of radioactivity.[1] This ultimately led to the formulation of the Einsteinian equation $E=mc^2$ and the production of the atomic bomb. This again brought nuclear physics and medicine together, for the bomb produced thousands of cases of injury from radiation. Prior to the explosion of the bomb in 1945, the cases of injury from radiation were confined to those cases of x-ray irradiation and to those persons who worked close to radium, such as watch dial painters. We have learned about radiation injury slowly and painfully. The first x-ray burn was reported in 1896, but it was not until 1915 that there was general recognition of the danger of x-ray.

As with any new discovery, there are those who propose it as a "cure-all," and so it was with x-ray irradiation and with radium. In the 1920's tonics were prepared from radium salts and sold as "cure-alls," and many people died subtle, lingering, painful deaths. Others believed that radiation was stimulating to the body, and many followed in their false beliefs and died.[2]

Unfortunately, for a period of time many doctors were not informed as to the dangers of mass radiation, but in 1945 the syndrome of acute radiation sickness became well known to a large portion of the medical and lay population of our country. Since 1945 much has been written about nuclear energy and the syndrome of acute radiation sickness.[3, 4, 7] It is our purpose to gather together some of the basic knowledge of this syndrome.

SIGNS, SYMPTOMS, AND DIAGNOSIS

The diagnosis of the injury sustained from a single exposure to whole body radiation arising from an external source will not be difficult if the history of exposure is known. However, in the absence of such a history, there are numerous other diagnoses which will occur to the physician as an explanation of the signs and symptoms. Correct diagnosis depends upon proper evaluation of three factors:[5]

1. Was the patient exposed to sufficient radiation?
2. The development of typical symptoms.
3. The occurrence and the time-trend of leukopenia and lymphopenia.

The mechanism of radiation injury is not known, but there are many theories and fragments of knowledge concerning its effect upon biological material. These will be discussed in a later section.

Two entities are considered when one studies the problem of radiation injury. One, commonly called "Radiation Disease," is that entity which follows chronic exposure or exposure of a limited area of an individual to therapeutic x-ray or radioactivity; the other, the one with which we are concerned, is called "Radiation Syndrome" and is composed of those signs and symptoms which follow acute, whole body, penetrating radiation exposure. As far as is known, these symptoms and signs are the same in pure radiation injury as in cases of radiation injury accompanied by burns or trauma.

These symptoms, in general, appear first in those more radiosensitive systems of the human body. There is no one symptom or laboratory test that can diagnose radiation syndrome, but there are certain specific and suggestive symptoms which combine into the complex known as the radiation syndrome. The earlier the appearance of these so-called specific or suggestive symptoms, the more severe the radiation injury.[5]

Dunham[5] lists the specific symptoms of radiation injury as epilation and purpura and the suggestive symptoms as vomiting on the day of exposure, oropharyngeal lesions, and hemorrhagic manifestations.

In the Japanese bombings only 5 to 15 per cent of the casualties were caused by ionizing radiation alone.

This is one of 11 theses, written by fourth year students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group judged to be the best by the faculty at the school.

Thesis written while the author was a fourth year student. Dr. van der Smissen is now interning at the United States Public Health Service Hospital, Baltimore, Maryland.



PROVED

mycin®

BRAND OF OXYTETRACYCLINE

rapid absorption

wide distribution

prompt response

excellent toleration

Pfizer

PFIZER LABORATORIES, *Brooklyn 6, N.Y.*

DIVISION, CHAS. PFIZER & CO., INC.

The radiations produced by these bombs consisted chiefly of neutrons and gamma rays. The clinical syndrome of uncomplicated radiation injury (i.e., pure radiation, no trauma or burns) in these bombings can be divided into four phases.[44]

PHASE 1

Onset—an hour or so after exposure.

Duration—a day or two.

Symptoms—prostration, shock-like states, nausea, vomiting, and sometimes diarrhea. Death may occur.

PHASE 2

A period of relative well-being between Phases 1 and 3. Patient may feel well enough to go back to work.

Duration—varies; the shorter it is, the greater the amount of exposure.

PHASE 3

Developing fever, diarrhea, epilation, hemorrhagic manifestations, and ulceration. It is the main part of the illness.

PHASE 4

Death, or prolonged convalescence.

Few of these symptoms are seen immediately; they take some time to develop. Other toxic symptoms develop immediately after or during radiation. These symptoms resemble shock, and there is considerable evidence that the greatest effect may be on the adrenal cortex. In this early phase there is dehydration, fall in blood pressure, tachycardia (which varies directly with the amount of radiation absorbed), dyspnea, and extreme prostration; other toxic symptoms are vomiting, nausea, diarrhea, and malaise. They last 24 to 48 hours, and then there is a latent period. The duration of this latent period depends upon the intensity of the dosage of radiation, being shorter when the dosage is greater. This is followed by more intense symptoms and new symptoms related to the hemopoietic system. Physiopathologic manifestations involving adrenal function, the adaptation syndrome, intoxication by histamine-like substances, and bacterial invasion also take place.

In the Japanese bombings epilation began during the second week after exposure, and regrowth started about the sixth week. In the survivors it was complete in four to five months. The cutaneous purpura usually did not appear until the third week. Besides the hemorrhagic manifestations other than purpura (epistaxis, menorrhagia, hematuria, metrorrhagia, melana, hemoptysis, and hematemesis), severe whole body radiation injury produced leukopenia, anemia, and thrombopenia.

Of the suggestive symptoms of radiation injury, the oropharyngeal lesions occurred prior to the 39th day. These were composed of dusky inflammation of the mucous membrane, soreness of the gums, and ulceronecrotic tissue breakdown.

There is a difference in reactions from various types of radiation. The radiation of the original Japanese bombings was soft, but that of the Los Alamos accidents was hard gamma radiation which presented a slightly different symptom complex, although basically both were the same syndrome.

In 1948 there were reported four cases of beta ray burn in personnel at Eniwetok. Their chief complaints were burning and itching. During the H-bomb tests on March 1, 1954, several fishermen were contaminated with radioactive debris. Their symptoms were somewhat different, starting with burning and itching, then gastrointestinal symptoms occurred, followed by blistering and progressing into the radiation syndrome Stage II or III (see chart on page 662).

The only cases of radiation syndrome in human beings are those which were the result of accidents (such as those cited by Hempelmann[7]), x-ray overdosage, or bombs. The dosage was not accurately known, nor were adequate controls run. One can only extrapolate from animal experiments as to the probable dosage ranges for the various types of radiation syndromes.

Some authors believe that an acute dose of 200 or more r will prove fatal to humans, whereas others set the minimum at 300 r; obviously the greater the dosage the greater the percentage of fatality. In acute total body radiation 400 r ∓ 100 r is considered the median lethal dose, the LD-50; and those of greater than 600 r are considered 100 per cent fatal, or LD-100.[5, 6, 10]

The radiation syndrome can be classified roughly into three groups, using the dosage and severity of symptoms as a basis: these are Group I (very severe), Group II (severe), and Group III (mild or moderate). Group I shows the only true primary radiation syndrome. The others start with the same symptoms, but other symptoms develop as a result of the ionization effect upon the hemopoietic system. These groupings are compared in the following chart.

Knowlton,[8] after extensive study of the value of blood counts in individuals exposed to ionizing radiation, concluded that, although the routine blood count is a poor detector of radiation injury in chronic radiation, it is still the best laboratory test available.[11] The lengthening of the prothrombin time also has been suggested as a means of detecting over-exposure to radiation.[12] There is no single laboratory procedure which is diagnostic, but the over-all blood picture shows pancytopenia and anemia. The symptoms of thrombocytopenic purpura and agranulocytosis also are seen.

It has been reported[7] that the anemia is of the macrocytic hyperchromic type.



for the 3 patients in 4



with seborrheic dermatitis of the scalp



Have you prescribed SELSUN *for them yet?* Here are the results you can expect: complete control in 81 to 87 per cent of all seborrheic dermatitis cases, and in 92 to 95 per cent of common dandruff cases. SELSUN keeps the scalp scale-free for *one to four weeks*—relieves itching and burning after only two or three applications.

SELSUN is applied and rinsed out while washing the hair. It takes little time, no complicated procedures or messy ointments. Ethically advertised and dispensed only on your prescription. In 4-fluidounce bottles.

Abbott



prescribe...

SELSUN®

SULFIDE *Suspension*

(Selenium Sulfide, Abbott)

405065

ACCEPTED COUNCIL ON PHARMACY AND CHEMISTRY

TYPES OF RADIATION SYNDROME AND THEIR SIGNS AND SYMPTOMS[5, 6]

GROUP I	GROUP II	GROUP III
Very severe cases	**Severe cases**	**Mild to moderate cases**
Primary type of radiation sickness.	Effects secondary to the effect of radiation on the hemopoietic system.	
LD-100 dosage. 600 r, or more, total body exposure (acute).	LD-50 dosage. About 400 r acute total body exposure	Less than LD-50 dosage. 200-300 r acute total body exposure.
	1st DAY	
Beginning in 1-2 hours: malaise, nausea, vomiting, asthenia, moderate diarrhea, and prostration. Rapidly developing and persistent leukopenia.	Beginning in a few hours, rarely continuing more than 24 hours: nausea, vomiting, diarrhea, lymphopenia.	Vomiting may occur in a few hours. Symptoms and signs not constant.
	NEXT FEW DAYS	
May go on to immediate exhaustion and death in a few days. Others have spontaneous remissions of 2-3 days with only malaise and then a return of symptoms with: sanguinous diarrhea, progressive sustained fever, thirst, emaciation, and extreme dehydration.	Second day shows lymphopenia of 800 lymphs/cu. mm.	No definite symptoms. Moderate lymphopenia.
	4th to 20th DAYS	
Death in 10-14 days, but may die within a few hours of exposure. Death in shock, exhaustion, coma, and delirium.	First a latent period of 8-10 days relatively free from symptoms and may even go back to work. May have some malaise and fatigability.	No definite symptoms for two weeks, then may have: epilation, malaise, sore throat, petechiae, diarrhea, or weight loss.
Purpura and epilation may appear shortly before death. Hemorrhagic manifestations do not seem to be responsible for fatal outcome.	Then, at any time up to the 20th day, the individual may become seriously ill, show any or all of the following symptoms: agranulocytosis, and/or thrombocytopenic purpura; epilation (scalp chiefly) at end of second week; nausea, vomiting, diarrhea, anorexia, and prostration; unremitting fever; infection and wound breakdown; severe leukopenia and general sepsis, melana, epistaxis, and hematuria; menorrhagia and metrorrhagia; petechiae, ecchymosis, and hemorrhage; ulcerative gastritis and enteritis; oropharyngeal ulcers.	
	In 20-40 days death occurs in 50 per cent of the untreated cases. Death is usually due to sepsis or with hemorrhagic manifestations dominating the picture.	

The rapidity of onset of the various hematologic findings is determined by the susceptibility of the various cells to radiation and by the intensity of radiation. The following is a table of the responses of the three groups of radiation.[10, 27]

THE HEMATOLOGIC REACTION

GROUP I	GROUP II	GROUP III
Lymphopenia (10 per cent of normal in first 30 hours).	Granulocytes drop to a few hundred.	Variable blood changes.
WBC falls to 2,000/cu. mm. and may drop to 200.	Platelets fall to 10,000/cu. mm.	Moderate leukopenia.
Red cells show little adverse effect as their life span is 120 days.	Agranulocytosis.	Moderate anemia.
Reticulocyte counts reflect the bone hypoplasia.	Thrombocytopenic purpura.	
Purpura and anemia are NOT characteristic.	Aplastic anemia after 4th-5th week.	
	Increased capillary fragility and heparin disturbance.	

The laboratory procedures are standard for the above determinations and are known to all. In general, the hematologic picture may indicate the prognosis and severity of the disease. If the total white blood count is less than 500/cu. mm., recovery is unlikely. The lymphocytes are even more indicative in that a total lymphocyte count of greater than 1,500/cu. mm. at 48 hours after exposure indicates that radiation injury of clinical significance is unlikely. On the other hand, a total lymphocyte count of less than 800 cells/ cu. mm. at 48 hours after exposure is presumptive evidence of severe radiation injury.

Total neutrophile counts drop more slowly, but if they reach less than 1,000 cells/cu. mm. within one week the outcome is usually fatal. With less severe doses, and less danger of fatality, the drop takes 10 to 20 days to reach this level.

The thrombocytopenia and the purpura develop more slowly. The thrombocytes remain stable for three to four days and then have a rapid drop, nearly to zero in the second week. They remain low for two to four weeks. This would indicate that the fault lies not in the circulating thrombocytes but in the development from the megakaryocytes of the bone marrow.

Anemia is the slowest to develop of the blood dyscrasias, and it is the most persistent. It takes two to three weeks to develop and attains its lowest point in about four to five weeks. As would be expected, any rapid drop in hemoglobin, red count, or hematocrit suggests rapid blood loss.

The reticulocyte count is one of the best indicators that the patient is recovering. The reticulocytes will be absent for almost three to four weeks, and then any rise is a good sign.

Gastrointestinal symptoms occur in two phases. The first is related to the radiation itself and is manifest by nausea and vomiting; the second is the bacterial overgrowth from the intestinal tract due to reduction

50 *and seven*

YEARS TREATING ALCOHOL AND DRUG ADDICTION

In 1897 Doctor B. B. Ralph developed methods of treating alcohol and narcotic addiction that, by the standards of the time, were conspicuous for success.

Twenty-five years ago experience had bettered the methods. Today with the advantages of collateral medicine, treatment is markedly further improved.

The Ralph Sanitarium provides personalized care in a quiet, homelike atmosphere. Dietetics, hydrotherapy and massage speed physical and emotional re-education. Cooperation with referring physicians. Write or phone.

The

RALPH

SANITARIUM

A Unit of the Benjamin Burroughs Ralph Foundation for Medical Research

Telephone VIctor 3624



529 HIGHLAND AVENUE • KANSAS CITY 6, MISSOURI

Ralph Emerson Duncan, M.D., Medical Director

of body defenses, and this is shown by sanguinous diarrhea and necrotizing ulcers.[6]

As is to be expected, deaths in the various groups are due to differing immediate causes. Those of Group I who die within a few hours of exposure do so from shock and exhaustion; those who linger a while go into coma and delirium, and then die. The exact cause of death is unknown, but probably it is due to the ionization affecting the enzyme systems.

Deaths in the severe cases (Group II) are usually due to:[5] (1) acute blood loss, (2) septicemia, and (3) hemorrhage into vital structures.

Those who die in Group III usually do so as a result of complications, not directly due to radiation. These complications most often affect the respiratory system and are usually fatal after the sixth week. In such cases one sees: (1) bronchiectasis, (2) chronic abscesses, (3) tuberculosis, and (4) refractory anemia.

Those 50 per cent in Group II who survive continue to have symptoms for weeks and months. A few instances of ocular cataract have been observed in survivors of the Japanese bombing, and in the Los Alamos accidents it was found that several of the exposed persons of relatively young age developed cataracts rather rapidly and that these closely resembled the senile type of cataract.

The early reaction seen in Groups I and II is the true radiation syndrome and is caused purely by the radiation absorbed and its ionization. Peculiarly, the intensity of these primary symptoms varies and is not correlated with radiation dosage received.[16] In both groups this initial reaction is followed by a latent period. The duration of this period depends upon the intensity of the dosage of radiation, being shorter with a greater dosage. In Group I the symptoms that return after the latent period are the same as those preceding, only more intense; those in Group II are slower coming on and are ordinarily of the nature of those evident in hemopoietic failure and bacterial invasion.

The full significance of late or delayed radiation effects is not known because of two factors: one, so few cases of radiation exposure have been studied, and two, insufficient time has elapsed since exposure to bring out the latent or genetic effects. Latent reactions usually develop years after exposure, and a latent interval of 25 to 30 years would not be considered uncommon.

Below are listed the possible latent effects of radiation:[44] (1) adult cataracts, (2) skin cancer at site of exposure, (3) leukemia, (4) aplastic anemia, (5) other forms of cancer, (6) developmental anomalies, (7) sterility, (8) shortening of life span, and (9) genetic mutations in germ cells.

THERAPY

To date there is no standard, specific treatment for this radiation syndrome. Practically all drugs in the pharmacopeia and all types of treatment known have at one time or another been proposed as therapy for radiation syndrome. Various vitamins, liver extracts, and insulin have been hailed as preventive or curative agents, but they have not yet been proved effective. Lacking a specific treatment, we must fall back on symptomatic therapy.

The basic principles of treatment are: (1) proper fluid and electrolyte balance, (2) adequate, good nutrition, (3) sedation as indicated and sufficient rest, (4) whole blood transfusions as indicated and treatment of hemorrhage and anemia, (5) antibiotics to stem septicemia, and (6) additional therapy for concomitant conditions such as burns or trauma.

The patients in Group II classification make up a group in which good medical treatment can either save or lose a life. Rarely will one save a patient from Group I.

Early use of saline and 5 per cent glucose will save time and effort in combating acid-base imbalance and dehydration brought about by diarrhea and vomiting. Once dehydration and acid-base imbalance have developed, the problem becomes more complicated.

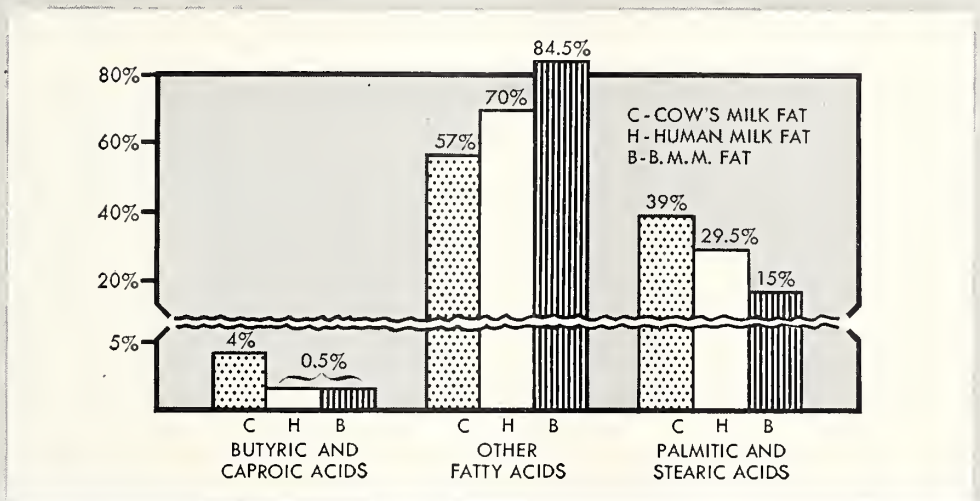
Adequate nutrition should include a bland low-residue high-calorie diet (3,000 to 4,000 calories per day) with high protein (150 to 200 grams per day). Both oral and parenteral routes must be employed. It is noted that 1,000 cc. of plasma (derived from 2 liters of blood) will contain 50 to 60 grams of complete, well balanced protein and represents about 250 calories.[13] One drawback is that the continued use of plasma tends to retard red cell production. To offset this, fresh whole blood should be used if possible. Large amounts of citrated blood will bind the blood calcium and cause hypocalcemic tetany. To forestall this, judicious use of calcium gluconate is indicated.

The use of many drugs and vitamins has been advised. Many so-called "specific" therapeutic measures have been advocated. Among these are sulfhydryl agents to reverse the enzyme inhibition; antihistamine compounds to neutralize toxins and released histamine-like compounds; adrenal hormones to replace losses; agents to maintain water and electrolyte balance; prophylactic antibiotics; the prevention and treatment of anemias and hemorrhagic tendencies; and means to prevent and remedy the marked loss of tissue and negative nitrogen balance.[9]

At one time antihistaminics were thought to be beneficial. It is now known that although antihistamine ointment may aid in protecting the skin during radiation therapy,[14] when taken internally antihistamines have a synergistic effect with radiation and increase its lethality in experimental animals. Antihistaminic drugs cause a pronounced leukocytosis in normal guinea pigs that receive the drug daily for periods of 14 to 23 days. Gallbladder rupture and

THE FATS IN... *Baker's Modified Milk*

...ARE READILY TOLERATED AND DIGESTED



● Baker's Modified Milk provides approximately 85% of its fatty acid composition in the more readily tolerated and digestible range (as shown in the fat chart above). This compares with 57% for cow's milk fat and 70% for the fat of human milk.

The fat composition of Baker's is only one of many reasons why this product is used so successfully in feeding the newborn term infant, the premature infant, and the older infant who does not "handle" butterfat.

Baker's is a high-quality* milk diet complete in all known essential nutrients.

★

*Made from Grade A milk (U. S. Public Health Service Milk Code) which has been modified by replacement of the milk fat with vegetable and animal fats and by the addition of carbohydrates, vitamins, and iron.

★

BAKER'S MODIFIED MILK

THE BAKER LABORATORIES INC.

Milk Products Exclusively for the Medical Profession

Main Office: Cleveland 3, Ohio
Plant: East Troy, Wisconsin

Division Offices: Atlanta, Dallas, Denver, Greensboro, N. C., Las Angeles, San Francisco, Seattle

ACCEPTED COUNCIL ON FOODS AND NUTRITION AMERICAN MEDICAL ASSOCIATION

enlargement of the common duct and gallbladder are observed in chronically medicated and irradiated guinea pigs. There is also a significant decrease in coagulation time in these animals.

There is no doubt of the value of blood transfusion in the initial shock phase, but what added advantage does it give? Allen[17] has shown that less severe irradiation anemia is the only benefit noted. Thus we assume that in other than the shock phase, it is well to reserve whole blood transfusions for those patients who show clinical bleeding, a white blood count of 1,000 or less, or a hemoglobin of less than 70 per cent. It is best to maintain hemoglobin at 10 grams or more.

Wide spectrum antibiotics (aureomycin, terramycin, etc.) seem to be valuable in combating infection, especially of certain gram-negative intestinal bacteria and in controlling diarrhea. The use of these antibiotics the day of exposure, continuing for 21 days, is to be recommended if the supply of antibiotics is adequate. Dosage is in the range of 500 mg. every six hours. Once severe symptoms have appeared, use both oral and parenteral antibiotics.

So far we have discussed therapeutic measures after exposure. There are many studies in experimental animals which indicate that certain prophylactic measures may be effective in combating the acute radiation syndrome. Prophylactic cysteine,[19] estrogens,[18] or glutathione[20] in tests with mice have significantly lowered the expected mortality of lethal dosage radiation. Oxygen deprivation or cyanide intoxication during exposure to lethal dosages of irradiation also reduced expected mortality. None of these agents are of value if administered after the radiation exposure has occurred.

Jacobson[23, 24, 25] has demonstrated that shielding the spleen of mice during exposure, or transplantation of as little as 10 mg. of fresh spleen to mice after exposure to lethal dosages of x-irradiation, increases survival and hastens regeneration of blood forming tissue and of the gastric lining and the intestinal tract.

This study strongly suggests the presence of a chemical substance in normal tissue capable of hastening regeneration and repair of irradiated tissue. Recent experiments of Cole, Fishler, and Bond[45] seem to bear this out. Straube[26] finds that the adrenals are apparently involved only secondarily as part of the organism's buffer response to the stress of irradiation. The rationale of employing adrenal corticoids to alter radiation mortality is therefore open to question.

Other substances such as the so-called anti-heparin materials (protamine and toluidine blue) and vitamin K (other than the natural vitamin K) have been of little value either as prophylactic or as post-exposure treatment.

PATHOLOGY OF RADIATION INJURY

What is the mechanism by which high-energy waves and particles damage living things? A large number of investigators have been working on this problem and find that the farther they go into it, the more complex it becomes. The syndrome is initiated by protoplasmic injury, through the following mechanisms: (1) enzyme inhibition, particularly sulfhydryl enzymes; (2) some alterations in osmotic properties and cell permeability; (3) generalized protein denaturation, and (4) an intoxication by histamine-like compounds supposedly liberated as a result of radiation.

There is a reciprocal relationship between the degree and reversibility of the enzyme inhibition, the protein denaturation, and the alterations in osmotic properties and cellular permeability. As the dosage increases, the degree of reversibility decreases, and the mortality rises.[9] Fundamentally these effects are due to radiation's ionizing action on the atoms and molecules of which the tissue is composed.

This ionization is based on several mechanisms: (1) the photoelectric effect in which one of the electrons of the atom is ejected; (2) the Compton effect based on the acceleration of a slow moving electron to higher velocity; (3) pair production to produce a positive and a negative electron; (4) direct strike of radiation on a nucleus causing nucleus acceleration, and (5) formation of a radioactive nucleus as the result of the absorption of a particle by the nucleus.

These mechanisms combine to damage the cell by one or both of two effects:[27] (1) radiation (particle or wave) can strike the nucleus or pass through the protein molecule denaturing it, and (2) by manufacturing reactive groups of atoms that pass through the cell wall and react with the enzyme systems.

These may affect cells in various ways. It may kill the cells outright, it may interfere with their growth, it may change their genes and heredity. The problem of acute radiation syndrome is concerned with the first two possibilities as causes of immediate symptoms. The effect upon the genes and heredity may be delayed several generations before it shows up in the cells as a mutation.

The most common effect, since cells are 70 per cent water, is the ionization of water to HOH^+ which breaks down to $H+$ and $OH-$ or OH. These can react together with HOH and O_2 to form H_2O_2 (hydrogen peroxide). This in turn breaks down into HOH and nascent oxygen, and the free radicals OH and HO_2. These agents are powerful oxidizing substances and can react with cellular material and interfere with its normal chemical activities. This action causes the disruption of the enzyme systems and vital functions of

Calm AND *Relaxed*

WITH **MEBARAL®**



The calming influence of Mebaral is eminently helpful in

- tension and anxiety states
- nervous symptoms of the menopause
- neurasthenia
- mild psychoses
- hysteria
- hyperthyroidism
- migraine
- pruritus
- hyperemesis nervosa
- hyperemesis gravidarum
- restlessness and irritability associated with pain or infection
- cardiovascular disorders
- allergies
- alcoholism

DOSAGE:

Adults—32 mg. to 0.1 Gm. (optimal 50 mg.), 3 or 4 times daily.

Children—16 to 32 mg., 3 or 4 times daily.

HOW SUPPLIED:

Tablets of 32 mg. (½ grain)
Tablets of 50 mg. (¾ grain)
Tablets of 0.1 Gm. (1½ grains)
Tablets of 0.2 Gm. (3 grains) scored for division

Winthrop-Stearns INC.
NEW YORK 18, N. Y. WINDSOR, ONT.

Mebaral, trademark reg. U. S. Pat. Off., brand of mephobarbital

most of the body cells, and death occurs promptly without much evidence of morphological change.[28] This is typical of the sudden deaths or deaths within a few hours of heavy acute radiation, but those with less heavy radiation show less effect and go on to various stages of necrosis and tissue reaction.

It is postulated[27] that these oxidizing agents have their greatest effect upon the sulfhydryl groups (SH) of the enzyme systems. By oxidizing this group they put the enzyme out of action. Putting the enzyme system out of action cuts off the respiratory function of the cell, and the cell dies. If the enzymes have received only a moderate dose of radiation, this oxidation may be reversed by appropriate compounds.

These same radiations may cause breaking of the chemical bonds of proteins, changing of nuclear material (possibly by its effect upon desoxypentosenucleic acid), and the formation of mutations. Tahmissian[29] learned that anabolic enzymes were found to be associated with the nucleus material and were more susceptible to radiation damage than catabolic enzymes; in a resting cell a nucleus was found to be necessary for the maintenance of the status quo. Pyknosis of the resting nucleus was found to depend upon x-ray dosage, state of metabolism, and time. Repression of irradiation damage to the cell could be obtained by subjecting the cells to sub-metabolic temperatures. As is to be expected, those cells most radiosensitive (those growing fastest) are the first affected by radiation.

Rugh[30] found that mitosis was abruptly inhibited in all cells which had not started the process. Those cells which were in mitosis at the time of irradiation were either able to complete the process or the nuclei and chromatin were so severely damaged that the typical intermitotic "resting cell" stage could not be achieved. He found in the salamander larva (Ambylstoma punctatum) that tissues most resistant to x-ray damage were cartilage and muscle, probably because they were the further developed. The tissue most sensitive to x-ray damage in the larva was that of the central nervous system and the related sense organs. This tissue was the most actively differentiating tissue at the time of irradiation.

Swartz,[31] in a study of humans after excessive radiation exposure, found the most striking biochemical changes are: (1) an increase in urine "corticosteroid-like substances," (2) a prolonged increase in the "adenosine triphosphate" phosphorus in the peripheral white blood cells of a patient given total body x-ray for the treatment of a metastasizing parotid tumor; (3) pronounced terminal increases in urinary coproporphyrin excretion, and (4) increased excretion of "urorosein fraction" pigments in severely ill patients given large doses of total body x-irradiation.

In his animal experiments he[31] found the following: (1) a drop in urinary coproporphyrin excretion followed by a terminal rise; (2) an increased excretion of fecal urobilinogen (indicative of increased red cell destruction); (3) terminal increases in excretion of urinary urobilinogen; (4) increased terminal excretion of kynurenic acid, and (5) increased excretion of uric acid and a decreased uricolytic index.

Other metabolic changes that have been noted[10] show changes in purine and fat metabolism, a biphasic action in carbohydrate metabolism (initial fall of blood sugar for 12 to 24 hours followed by a rise), a rise in respiratory quotient, and a biphasic acid-base action (initial acidosis lasting about 72 hours followed by alkalosis).

Several authors[10, 32] list the radiosensitivity of tissues in the following descending order: (1) lymphoid tissue and bone marrow; (2) epithelial cells (reproductive organs, skin, mucous membrane); (3) endothelial cells; (4) connective tissue; (5) muscle and bone cells, and (6) nerve cells.

Lymphoid Tissue and Bone Marrow: Lymphoid tissue is more sensitive to radiation than is bone marrow, but the more rapidly reproducing lymphoid tissue bounces back faster than does bone marrow. Although the stem cells of both lymphoid tissue and bone marrow appear the same, it would seem from this that the stem cell of lymphoid tissue is more hardy. The lymphocytes are the first cells to show signs of regeneration, as indicated by the increase in their number in the blood. Lymphoid tissue injured by radiation tends to become edematous. These swellings, especially in the throat, ulcerate to become ulcerations common in the oropharyngeal phase of radiation syndrome. The lymphoid tissue in the intestinal tract also swells, producing sanguineous diarrhea, nausea, and vomiting.

Bone marrow changes are mirrored in those changes already mentioned for peripheral blood. They show a type of hypoplasia of the bone marrow. In vitro experiments with both animal and human red cells showed that alpha irradiation caused shrinkage, then swelling, and finally hemolysis. Local irradiation of a part of a single cell resulted in shrinkage of only the irradiated portion. This was followed by swelling of the whole cell and hemolysis.[32]

Ingram,[33] in studying the blood picture of cyclotron workers, found two types of unusual cells. These are early mononuclear cells and bi-lobed or double nuclei lymphocytes. The "early mononuclear cells" are large, peroxidase negative, have deeply basophilic cytoplasm and no nuclei. The "lymphocytes with bi-lobed nuclei" tend to be somewhat larger than typical large lymphocytes with the nuclei stain slightly lighter than the nuclei of the other lymphocytes, but they are similar in consistency and have no nucleoli. The cytoplasm is clear, basophilic, peroxidase negative, and commonly contains a few typical azurophilic granules.



Thank you doctor for telling mother about...



- The Best Tasting Aspirin you can prescribe
- The Flavor Remains Stable down to the last tablet
- 15¢ Bottle of 24 tablets (2½ grs. each)

We will be pleased to send samples on request

THE BAYER COMPANY DIVISION of Sterling Drug Inc., 1450 Broadway, New York 18, N. Y.

But both these cells have on occasion been found in workers not associated with the cyclotron project or with any other form of nuclear project.

Marks[34] states, "The most sensitive cellular constituent of the peripheral blood appears to be the lymphocyte, and the reduction in its values closely parallels that of the total number of leukocytes per cu. mm. The morphological changes in the lymphocytes of the peripheral blood after acute total body x-irradiation agree essentially with those seen upon histological examination of the lymphatic tissue in sacrificed animals. With semi-lethal doses, and to a lesser extent after doses below this value, mitosis ceases within an hour and destruction and phagocytosis of lymphatic cells, mainly small and medium lymphocytes, proceeds. 'Blast like' cells are occasionally seen with nuclei which appear to be dissolving. Other lymphocytes appear to have split nuclei and nuclei with considerable fragmentation. During the phase of regeneration in the bone marrow the hetrophiles in the circulating blood of rabbits show a shift to the left following acute total body x-irradiation. Mitrachondrial changes are variable."

Suter[35] also concurs in these various responses of the hematopoietic system to x-irradiation.

Epithelial Cells: The testes showed linings of abnormally large Sertoli cells and hyalinization of the basement membrane. Robinson,[36] in a case of the human testes in which the accurate radiation dosage is not known but exposure was only a fraction of a second to neutron bombardment, found that in three weeks after injury there was no evidence of spermatozoa, and the loss of facial and head hair had occurred. By the 12th to 15th month the spermatogenesis had begun to return.

A rare case is reported[37] in which a male had been sterile for 10 years following irradiation with x-rays and radium as treatment for carcinoma of the rectum. The birth of a daughter to his family indicated the regeneration of the damaged gonadal tissue. It was found on examination that both the child and the father were normal in every respect.

The changes in the ovary are not as striking as those in the testis. There is a change in the ova and also in the menstrual cycle. It is reported[38] that a dose of 2,400 mg. hr. of radium caused amenorrhea for four months, and then the patient became pregnant. There is also an increased incidence of miscarriages and premature births and an increased death rate among expectant mothers.

Pohle[39] found no significant difference in the healing of wounds in the skin in mild or moderate irradiation. Woodburne,[40] in a study of 100 patients receiving irradiation, found such general skin effects as erythema, edema, hyperpigmentation, dryness, atrophic and depigmented skin. Severe cases involving vessels had obliterative vasculitis and consequent ulceration. They urge surgery as soon as feasible for these latter cases. The incidence of epilation has already been mentioned. This epilation seemed to follow the course of ordinary baldness, first involving the front, then the top and back of the head.

Connective Tissue: Jolles,[41] using a sieve (or Chess) method of study, found that the connective tissue reaction takes place not only in irradiated tissue but also in surrounding tissue. The reaction consists of a deposition of an excessive amount of collagenous fibrils which are organized into solid coarse bundles in the exposed parts. Lymphatic channels and blood capillaries are compressed and blocked.

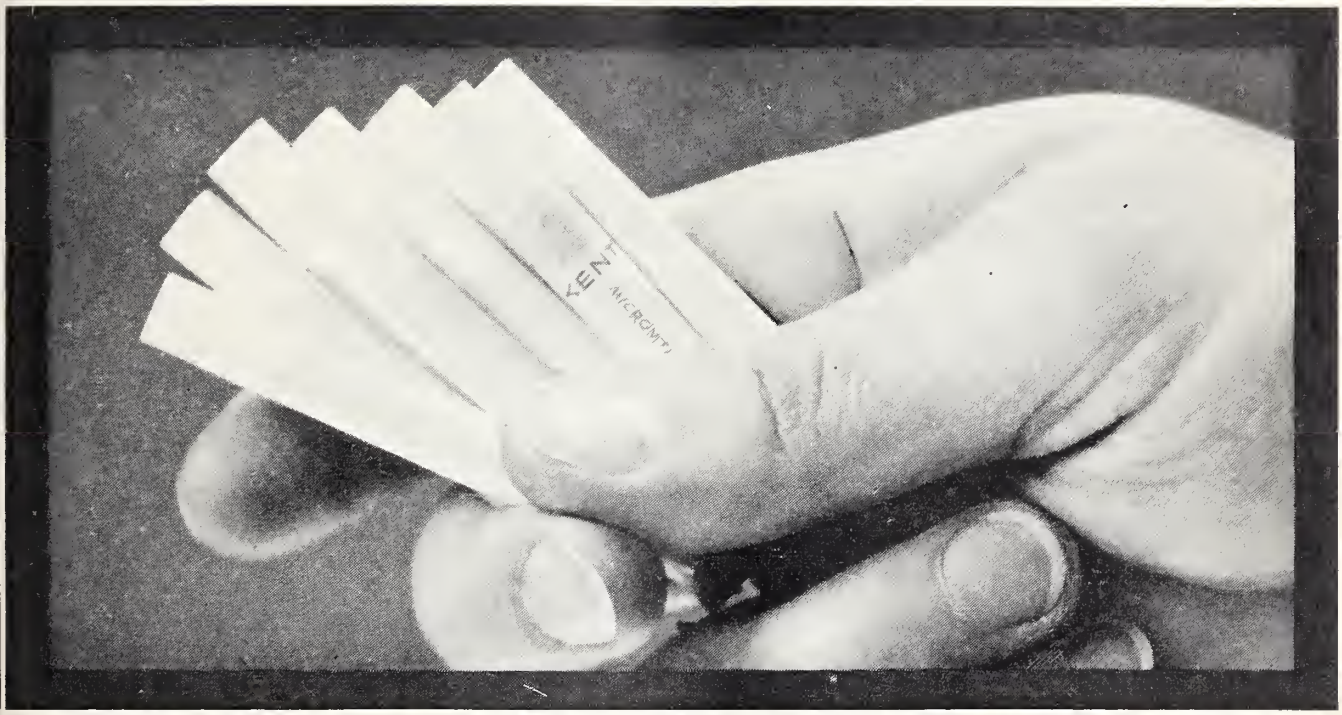
Nerve Tissue: Russel et al.[42] found, on irradiating rabbit brains, that areas of hemorrhage and necrosis formed and that the neurones were only focally and secondarily affected; the meninges and their blood vessels were unaffected; the "superficial parts of the cerebrum suffered no damage, although the cerebellar cortex was in some cases extensively damaged."

A very interesting study[43] was made comparing the physical growth and development of children who survived the Hiroshima and Nagasaki bombing with other Japanese children of the same age. In 1947 the exposed boys were shorter and lighter than the non-exposed boys, but there was slight difference in the exposed and non-exposed girls. Two years later there was no detectable difference between the two groups of girls, but the exposed boys were still shorter and lighter than the unexposed. A year later there was no significant over-all difference. These differences may be attributed to the consequences of total annihilation of the economy of the bombed areas rather than direct effect of the radiation. It may take several generations for the appearance of any mutations which may have occurred. It is highly probable that any mutations which formed would be lethal characteristics and therefore would not be propagated.

BIBLIOGRAPHY

1. Dietz, David: The story of science, Dodd, Mead, & Co., New York, pp. 193-201, 1942.
2. Sixth Semi-Annual Report of the AEC, July 1949, pp. 15-19, U. S. Government Printing Office, Washington, D. C.
3. Goldsmith, H. H.: Bibliography on radiation protection, Nucleonics 4:62-69 (June) 1949.
4. Painter, E. E., and Brues, A. M.: The radiation syndrome, New Eng. J. Med. 240:871-876 (June 2) 1949.
5. Dunham, C. L.; Cronkite, E. P.; LeRoy, G. V., and Warren, S.: Atomic bomb injury: radiation, medical aspects of civil defense. (Series of Special Articles Reprinted from the J.A.M.A.).
6. Sears, T. P.: The physician in atomic defense, Year Book Publishers, Inc., Chicago, pp. 150-157, 1953.
7. Hempelmann, L. H.; Lisco, H., and Hoffman, J. G.: The acute radiation syndrome: a study of nine cases and a review of the problem, Ann. Int. Med. 36:279-510 (Feb., part 1) 1952.
8. Knowlton, N. P., Jr.: The value of blood counts in individuals exposed to ionizing radiation, AECU-397, 25 pp.
9. Cronkite, E. P.: Pathogenesis and therapy of radiation illness, Med. Ann. Dis. Columbia 18:239-242 (May) 1949.
10. Draeger, R. H.: Therapy of acute radiation illness, radiological defense, Armed Forces Special Weapons Project, 1949.
11. Wilson, Rex H.: Ionizing radiation, Ohio State Med. J. 45:579-81 (June) 1949.
12. Ho. T. J.: Roentgen ray protection and irradiation hazards due to overexposure, X-ray Technician 20:353-355 (May) 1949.
13. Bauer, A. J.: Treatment of acute radiation illness, radiological defense, Armed Forces Special Weapons Project, 1949.

Why is it, Doctor, that one filter cigarette gives so much more protection than any other?



The answer is simply this: Among today's nine brands of filter cigarettes, KENT, and KENT alone, has the *Micronite Filter*... made of a pure, dust-free material that is so safe, so effective it has been selected to help filter the air in hospital operating rooms.

> In continuing and repeated impartial scientific tests, KENT's Micronite Filter consistently proves that it takes out *more* nicotine and tars than *any* other filter cigarette, old or new.

And yet, with all its superior protection, KENT's Micronite Filter lets smokers enjoy the full, satisfying flavor of fine, mellow tobaccos.

For these reasons, Doctor, shouldn't KENT be the choice of those who want the minimum of nicotine and tars in their cigarette smoke?



Kent

... the only cigarette with the MICRONITE FILTER

for the greatest protection in cigarette history

"KENT" AND "MICRONITE" ARE REGISTERED TRADEMARKS OF P. LORILLARD COMPANY

14. Mains, M. P.: Anti-histamine ointments for skin protection in radiation therapy, Radiology 52:579-581 (April) 1949.
15. Haley, T. J., and Harris, H.: The effect of several antihistaminic drugs on mortality due to x-irradiation, AECU-362 (July 8) 1949.
16. Ellis, F.: Volume dose in radiotherapy, Brit. J. Radiology 18:240, 1945 (Reviewed in Hempelmann, et al.).
17. Allen, J. G.; Moulder, P. V., and Enerson, E. M.: Irradiation damage and blood transfusion, Science 115-523 (May 9) 1952.
18. Patt, H. M.; Straube, R. L.; Tyree, E. B.; Swift, M. N., and Smith, D. E.: Influence of estrogens on the acute x-irradiation syndrome, AECU-524 (UAC-92).
19. Patt, H. M.; Tyree, E. B.; Straube, R. L., and Smith, D. E.: Cysteine protection against irradiation, Science 110:213, 1949.
20. Cronkite, E. P.; Breecher, G., and Chapman, W. H.: Mechanism of protective action of glutathione against whole body irradiation, Proc. Soc. Biol. and Med. 76:396, 1951.
21. Dowdy, A. H.; Bennett, L. R., and Chastain, S. M.: Protective action of anoxia against total-body roentgen irradiation of mammals, Radiology 55:879, 1950.
22. Bacq, A. H.; Herva, A.; Recomte, J., and Fisher, P.: Cyanide protection against x-irradiation, Science 111:356, 1950.
23. Jacobson, L. O.; Simmons, E. L.; Marcs, E. K.; Robson, M. J.; Bethard, W. F., and Gaston, E. O.: The role of the spleen in radiation injury and recovery, J. Lab. & Clin. Med. 35:746, 1950.
24. Jacobson, L. O.; Marcs, E. K.; Robson, M., and Zirkle, R. E.: The effect of spleen protection on mortality following x-irradiation, AECU-535 (UAC-111).
25. Jacobson, L. O.; Marks, E. L.; Gaston, E. O.; Robson, M., and Zirkle, R. E.: The role of the spleen in radiation injury, Proc. Soc. Exptl. Biol. & Med. 70:740-42 (April) 1949.
26. Struabe, R. L.; Patt, H. M.; Tyree, E. B., and Smith, D. E.: Influence of the level of adrenal cortical steroids on sensitivity of mice to x-irradiation, AECU-529 (UAC-102).
27. Spoerl, E.: The lethal effects of radiation, Sc. Am. (Dec.) 1951.
28. Warren, S.: The pathogenesis of irradiation sickness, Chicago Med. Soc. Bull. 51:957-9 (May) 1949.
29. Tahmissian, T. N.: The effect of x-radiation on the metabolic processes of the resting cell, J. Exptl. Zool. 112:449-63 (Dec.) 1949 (Nuclear Science Abstracts, 1950).
30. Rugh, R.: Histological effect on the embryo following x-radiation, AECD-2596, 1949.
31. Schwartz, S.: Some biochemical effects of ionizing radiations, Bull. Univ. Minn. Hos. & Minn. Med. Found. 20:617-654 (June) 1949.
32. Buchsbaum, Ralph, and Zirkle, R. E.: Shrinking and swelling after alpha irradiation of various parts of large erythrocytes, AECU-501 (UAC-114).
33. Ingram, M., and Barnes, S. W.: Observations of the blood of cyclotron workers, AECU-334.
34. Marks, E. K.: Effects of radiation exposure on the cells of the peripheral blood, MDDC-912, 1947.
35. Suter, G. M.: Response of hematopoietic system to x-rays, MDDC-824, 1947.
36. Robinson, J. N., and Engle, E. T.: Effect of neutron radiation on the human testes: a case report, J. Urol. 61:781-4 (April) 1949.
37. Korbler, J.: Concerning healthy offspring after radium radiation of the male gonads, Radiologia Clin. 18:240-4 (July) 1949 (Nuclear Sc. Abst.-1950).
38. Giles, A. M.: Pregnancy following pelvic irradiation, J. Ob. Gyn. Brit. Empire 56:1041-3 (Dec.) 1949.
39. Pohle, E. A.; Ritchie, G., and Moir, W. W.: Studies of the effect of roentgen rays on healing of wounds. III. Histological changes in skin wounds in rats following postoperative irradiation with very small and moderate doses, Radiology 52:707-14 (May) 1949.
40. Woodburne, A. R., and Philpott, O. S.: Radiation effects on the skin and their treatment, J. Mich. State Med. Soc., 48:461-79 (April) 1949.
41. Jolles, B.: The study of connective-tissue reaction to radiation: The sieve or Chess method, Brit. J. Cancer 3:27-31 (March) 1949.
42. Russell, D. S.; Wilson, C. W., and Tansley, K.: Experimental radionecrosis of the brain in rabbits, J. Neurol. Neurosurg. Psychiatry 12:187-95 (August) 1949.
43. Gruelich, W. W.; Crismon, C. S., and Turner, M. L.: The physical growth and development of children who survived the atomic bombing of Hiroshima or Nagasaki, J. Ped. 43:121-145, 1953.
44. Hemplemann, L. H., and Hoffman, J. G.: Practical aspects of radiation injury, Annual Rev. Nuclear Sci. Vol. III, 1953, Ann. Rev. Inc., Stanford, pp. 369-392.
45. Cole, L. J.; Fishler, M. C., and Bond, V. P.: Post-irradiation protection activity associated with spleen sub-cellular components, U. S. Naval Radiological Defense Laboratory 378 (1952).

Thirty-one medical films shown at the recent A.M.A. session in San Francisco were viewed by 8,993 persons.

A total of 12,063 physicians registered at the June meeting of the A.M.A. in San Francisco. The total registration figure was 42,969.

HEARING *is their business!*

These are the Audivox Hearing Aid Dealers who serve you in Kansas. Audivox dealers are chosen for their competence and their interest in your patients' hearing problems.

TOPEKA

The Topeka Hearing Center
824 Quincy Street
Tel: 4-9609

WICHITA

Kansas Hearing Center
223 South Broadway
Tel: 4-8614

KANSAS CITY, MISSOURI

Southwestern Hearing Aid Service
202-12th and Walnut Building
Tel: Harrison 0541

ST. JOSEPH, MISSOURI

Southwestern Hearing Aid Company
216 North Seventh Street
Tel: 4-8515

HASTINGS, NEBRASKA

O. E. Kullberg
110 Forest Boulevard

audivox
TRADE-MARK
SUCCESSOR TO
Western Electric
HEARING AID DIVISION



Audivox new all-transistor model 71 hearing aid

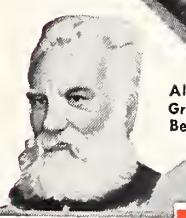
pedigree

Only a flawless pedigree — a long and illustrious ancestry of purebreds — can produce a champion show dog.

Only **audivox** in the hearing aid field can trace an ancestry that includes both Western Electric and Bell Telephone Laboratories. **audivox** lineage springs from the pioneer experiments of Dr. Alexander Graham Bell, which were furthered by the development of the hearing aid at Bell Telephone Laboratories, brought to fruition by Western Electric and **audivox** engineers.

Pedigreed in its field, **audivox** successor to Western Electric Hearing Aid Division, brings the boon of better hearing, and its enrichment of living, to thousands. With the magical modern transistor, with scientific hearing measurement and scientific instrument-fitting, serviced by a nation-wide network of professionally-skilled dealers, **audivox** moves forward today in a proud tradition.

TO THE DOCTOR: Send your patient with a hearing problem to a career Audivox and Micronic dealer, chosen for his interest, integrity and ability. There is such an Audivox dealer in every major city from coast to coast.



Alexander Graham Bell

audivox

Successor to *Western Electric* Hearing Aid Division

123 Worcester St., Boston, Mass.
The Pedigreed Hearing Aid

ANNOUNCEMENTS

The seventh annual convention of the International Academy of Proctology will be held at the Plaza Hotel, New York City, March 23-26, 1955. The program will give special emphasis to anorectal presentations and panel discussions. All physicians are invited to attend, regardless of affiliation with the Academy. There is no attendance fee.

The ninth annual seminar in ophthalmology and otolaryngology of the University of Florida will be held at the Sans Souci Hotel, Miami Beach, January 17-22. Lectures on ophthalmology will be presented during the first three days, those on otolaryngology during the last three days.

The Mississippi Valley Medical Society has announced its 1955 essay contest. A cash award of $100, a gold medal, and a certificate of award will be given the author of the best unpublished essay on any subject of general medical interest and practical value. Contestants must be residents of the United States and members of the American Medical Association. Closing date for the contest is May 1, 1955. Details may be secured from Dr. Harold Swanberg, secretary, 209 W.C.U. Building, Quincy, Illinois.

The next examinations for the American Board of Physical Medicine and Rehabilitation will be held in Philadelphia, June 5 and 6, 1955. Final date for filing applications is March 1, 1955. Applications for eligibility should be mailed to the secretary, Dr. Earl C. Elkins, 30 North Michigan Avenue, Chicago 2, Illinois.

A new short course offering expert instruction on the correct way of utilizing "Standard Nomenclature of Diseases and Operations" will be offered at A.M.A. headquarters, Chicago, February 7, 8, and 9. The program will be divided into three parts: (1) lectures covering basic principles, construction, and installation, plus discussion on the tumor and operation sections and the handling of specific problems; (2) anatomy as it pertains to the topographic section, and (3) practice in coding. Registration will be limited to 150.

The Council on Scientific Assembly, American Medical Association, has announced deadlines for

ANNUAL CLINICAL CONFERENCE

CHICAGO MEDICAL SOCIETY

March 1, 2, 3, 4, 1955

Palmer House, Chicago

DAILY HALF-HOUR LECTURES BY OUTSTANDING TEACHERS AND SPEAKERS on subjects of interest to both general practitioner and specialist.

PANELS ON TIMELY TOPICS.

MEDICAL COLOR TELECASTS.

TEACHING DEMONSTRATIONS.

SCIENTIFIC EXHIBITS worthy of real study and helpful and time-saving TECHNICAL EXHIBITS.

The CHICAGO MEDICAL SOCIETY ANNUAL CLINICAL CONFERENCE should be a MUST on the calendar of every physician. Plan now to attend and make your reservation at the Palmer House.

..in 2 hours or less

Filmtab*

Erythrocin® Stearate

(Erythromycin Stearate, Abbott)

disintegrates faster than enteric-coated erythromycin

TISSUE-THIN FILMTAB COATING (marketed only by Abbott) actually starts to dissolve within 30 seconds after administration —makes ERYTHROCIN available for immediate absorption. Tests show that new Stearate form definitely protects ERYTHROCIN from gastric juices.

BECAUSE THERE'S NO DELAY FROM AN ENTERIC COATING, your patient gets high, inhibitory blood levels within 2 hours—instead of 4-6 as before. Peak concentration at 4 hours, with significant levels for 8 hours.

USE FILMTAB ERYTHROCIN STEARATE against the cocci . . . and especially when the organism is resistant to other antibiotics. Low in toxicity—*it's less likely to alter normal intestinal flora than most oral antibiotics.* Conveniently sized (100, 200 mg.) in bottles of 25 and 100. Abbott

*TM for Abbott's film sealed tablets, pat. applied for

410203

those who wish to participate in the 1955 meeting of the A.M.A. at Atlantic City. The deadline for those who wish to present section papers is December 15, 1954; for those who wish to present scientific exhibits, January 10. Complete information may be obtained from the Council, A.M.A., 535 North Dearborn Street, Chicago 10, Illinois.

The next scheduled examination (Part I), written examination and review of case histories, for all candidates of the American Board of Obstetrics and Gynecology will be held in various cities on Friday, February 4, 1955. Twenty case abstracts are to be sent to the secretary by each candidate as soon as he receives notification of eligibility to take the Part I examination.

The Sixth American Congress on Obstetrics and Gynecology will be held at the Palmer House, Chicago, December 13-17, under the sponsorship of the American Committee on Maternal Welfare, Inc., and the American Academy of Obstetrics and Gynecology. The program will include 30 formal papers, 22 symposia and panels, luncheon discussion groups, and round-table discussions.

CLASSIFIED ADVERTISEMENTS

FOR SALE—Modern well equipped air conditioned eye, ear, nose, and throat clinic located in red grapefruit section Rio Grande valley, Texas. Location ideal. Will sell equipment separate if wanted. Cheap. For details write Journal 22-54.

GOVERNMENT INSTITUTION in the Kansas City area needs fulltime physician for general hospital and clinic practice. Suitable either for a physician seeking career in government service or possibly a semi-retired person wishing to return temporarily to clinical activity. Write the Journal 23-54.

FOR SALE—Castle autoclave size 8 inches by 16 inches, practically new, priced right. Write the Journal 19-54.

WILL GIVE most all office equipment and furnishing to a younger doctor who will locate in my town of 3,500 population for general practice. Retiring because of age. Write the Journal 20-54.

LABORATORY TECHNICIAN, registered, wanted for midwestern medical group of eight. Excellent facilities and liberal salary. Write the Journal 21-54.

FOR SALE—EENT practice and office equipment. Established 34 years in college town. Leaving because of wife's health—Write the Journal, 16-54.

FOR SALE—General Practice in small north central Kansas community, office and office equipment available. Residence for rent. Write the Journal, 17-54.

FOR SALE, Due to business change:
1 Brown-Burger Operating and Catheterizing Cystoscope—Excellent.
1 Infant Brown-Burger Single Catheterizing Cystoscope—New Condition.
1 Ophthalmoscope and Can—New.
1 Large Burglar Proof Bank Safe—Double Time Lock and Day Combination.
200 K.V. Westinghouse X-ray Machine, Rotary Anode Tube, Spot Film—Excellent.
Reply to the Journal, 18-54.

Changes of address should be reported to The Kansas Medical Society, 315 West Fourth Street, Topeka, Kansas.

UNIVERSITY OF KANSAS SCHOOL OF MEDICINE

Postgraduate Medical Courses

INDUSTRIAL AND OCCUPATIONAL MEDICINE
December 6, 7 & 8, 1954

Guest Instructors:

JAMES L. BEAVER, M.D., Assistant Medical Director, Boeing Airplane Co., Wichita.
MAC F. CAHAL, D.J., Executive Secretary, A.A.G.P., Kansas City.
E. H. CARLETON, M.D., General Medical Director, Inland Steel Co.; Member, Visiting Committee, Harvard University School of Public Health, Boston.
F. L. FEIERABEND, M.D., President, Board of Directors, Blue Shield, Kansas City.
R. EMMET KELLY, M.D., Instructor in Medicine, St. Louis University School of Medicine; Medical Director, Monsanto Chemical Co., St. Louis.
JOHN L. MORGAN, M.D., Internist, Emporia.
J. E. MOSIER, D.V.M., Professor of Veterinary Medicine, School of Veterinary Medicine, Kansas State College, Manhattan.
CHARLES NYBERG, M.B.A., Executive Assistant, A.A.G.P., Kansas City.
CHARLES R. ROMBOLD, M.D., Chief, Orthopedics Section, The Wichita Clinic, Wichita.
HARLAN R. SCHUYLER, Ph.D., President, Conservation Industries, Kansas City.
WILLIAM J. SHAW, M.D., Chairman, A.A.G.P. Commission on Education; Director, Lee Hospital, Fayette; President, Missouri State Board of Medical Examiners.
CHARLES F. SHOOK, M.D., Medical Director, Owens-Illinois Glass Co., Toledo; Member, Joint Committee on Industrial Health of the A.M.A., the A.A.G.P. and the Industrial Medical Ass'n.
H. SCOTT VAN ORDSTRAND, M.D., Chief, Department of Pulmonary Disease, The Cleveland Clinic, Cleveland.
VINCENT T. WILLIAMS, M.D., Medical Director, Fisher Body, Kansas City Division, and Sheffield Steel Corp., Kansas City.

SURGERY
January 17, 18, 19 & 20, 1955

Guest Instructors:

RICHARD B. CATTELL, M.D., Director, The Lahey Clinic, Boston.
DAVID M. DAVIS, M.D., Emeritus Professor of Urology, Jefferson Medical College of Philadelphia.
J. ENGLEBERT DUNPHY, M.D., Clinical Professor of Surgery, Harvard University Medical School, Boston.
DAVITT A. FELDER, M.D., Clinical Professor of Surgery, University of Minnesota School of Medicine, Minneapolis.
JOHN H. GRINDLAY, M.D., Section of Surgical Research, The Mayo Clinic; Associate Professor of Surgery, University of Minnesota Graduate School of Medicine, Rochester.
CHARLES HUFNAGEL, M.D., Associate Professor of Surgery and Professor of Surgical Research, Georgetown University School of Medicine, Washington.
AMOS R. KOONTZ, M.D., Assistant Professor of Surgery, Johns Hopkins University School of Medicine, Baltimore.
CHAMP LYONS, M.D., Professor of Surgery and Chairman of Department, Medical College of Alabama, Birmingham.
EARLE B. MAHONEY, M.D., Associate Professor of Surgery, University of Rochester School of Medicine, Rochester.
K. ALVIN MERENDINO, M.D., Associate Professor of Surgery and Director, Experimental Surgical Laboratories, University of Washington School of Medicine, Seattle.
H. WILLIAM SCOTT, JR., M.D., Professor of Surgery and Chairman of Department, Vanderbilt University School of Medicine, Nashville.
DANELY P. SLAUGHTER, M.D., Associate Professor of Surgery and Director, Tumor Clinic, University of Illinois College of Medicine, Chicago.
HARVEY B. STONE, M.D., Emeritus Associate Professor of Surgery, Johns Hopkins University School of Medicine, Baltimore.
ORVAR SWENSON, M.D., Clinical Professor of Pediatric Surgery, Tufts College Medical School, Boston.
HOWARD ULFELDER, M.D., Assistant Clinical Professor of Gynecology, Harvard University Medical School, Boston.
NATHAN A. WOMACK, M.D., Professor of Surgery, and Chairman of Department, University of North Carolina School of Medicine, Chapel Hill.

THE SOUTHARD SCHOOL

Intensive individual psychotherapy in a residential school, for children of elementary school age with emotional and behavior problems.

THE MENNINGER CHILDREN'S CLINIC

Outpatient psychiatric and neurologic evaluation and consultation for infants and children to eighteen years.

Department of Child Psychiatry

THE MENNINGER FOUNDATION

J. Cotter Hirschberg, M.D., Director Topeka, Kansas; Telephone 3-6494

★ PETRO'S SURGICAL APPLIANCES ★

Surgical Support Fittings by Trained Technicians.

Since 1930, Quality Appliances for Men, Women and Children.

- Abdominal Supports
- Back Supports
- Bras—Breast Forms
- Elastic Hosiery
- Knee Braces
- Ankle Braces
- Taylor Braces
- Trusses

"All Fittings Subject to the Doctor's Approval"

618-20 Quincy TOPEKA, KANSAS Phone 40207

THE MEDICAL PROTECTIVE COMPANY
FORT WAYNE, INDIANA

PROFESSIONAL PROTECTION EXCLUSIVELY SINCE 1899

specialized service assures *"know-how"*

TOPEKA Office:
J. E. McCurdy, Rep.,
1035 Randolph Avenue,
Telephone 2-3027

A Mutual, Open-end, Diversified Investment Fund with Full Management Discretion

MUTUAL TRUST

Offered by

MUTUAL DISTRIBUTORS, Inc.
1016 Baltimore • Kansas City 5, Mo.

Principal Underwriter and Distributor

PROSPECTUS ON REQUEST

PLEASE SEND ME PROSPECTUS

*Name*_____

*Address*_____

*City*_____ *State*_____

Clarence W. Glassen, State Representative

BOOK REVIEWS

Living with a Disability. By Howard A. Rusk and Eugene J. Taylor. Published by the Blakiston Company, Inc., New York. 207 pages, 275 illustrations. Price $3.50.

This unique book was written by one of the nation's leading authorities on medical rehabilitation in collaboration with a noted lay lecturer in the field of rehabilitation and devices for the handicapped. With the impetus given the rehabilitation concept by the nation-wide interest in the physically handicapped during and following World War II, it was evident to the authors that data relative to devices useful in disabilities must be assembled.

The initial study, the Self-Help Device Research Project, resulted in the publication of several pamphlets in which the fabrication of devices of value in certain types of disability was described. A second study was concerned with evaluation of individual capabilities and limitations in specific disabilities. The present book is the outgrowth of these projects with reference particularly to time-proved devices and suggestions, together with sound advice concerning the psychology of disability and the philosophy of living of those so disabled.

The initial portion of the book concerns the correct assessment of a physical disability. This is measured by a multiplicity of commonplace functions aptly termed "Activities of Daily Living." The clever arrangement of these tests soon narrows the disability to the actual areas in which there is real handicap and in which, therefore, attention must be concentrated in the rehabilitation sense. Sound advice is given regarding the relative advantages and disadvantages of the agencies from which assistive devices may be fabricated whether it be metal, wood, plastic, wire, leather, etc. The devices rather naturally fall into several categories: those concerned with eating, with dressing and physical appearance in general, with bathing and other toilet functions (including valuable sketches of bathroom plans for the wheelchair patient), with writing and similar hand activities, with transportation facilities in the sense of wheelchairs, walkers, and crutches, with kitchen activities for the housewife, with planning the home around the physical disability, with hobbies and relaxation, and finally with those devices useful in keeping the workman on his job. Narration is clear and concise and is certainly held to a minimum. The entire book is profusely illustrated, which is one of the reasons why narration is so conservatively employed.

This book is so unique that it may well become a classic in its field. Physicians who, to state the

It's Convenient to Buy Kanox

Send your patients to our sickroom department for all their needs. You may rest assured they will receive good courteous service at a reasonable price.

Walkers	Sales	Canes & Crutches
Wheelchairs	&	Commodes
Beds	Rentals	Supply Items

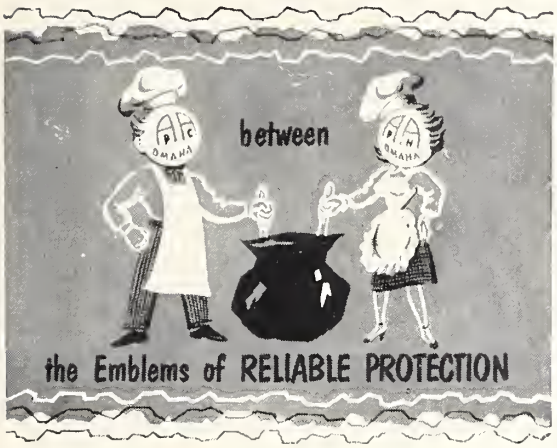
We are now stocking many and varied items used in hospitals and doctor offices. Would you give us an opportunity to service your account? Every article sold with an unconditional guarantee of satisfaction. Why not try us?

KANSAS OXYGEN, Inc.

Hospital Dept.

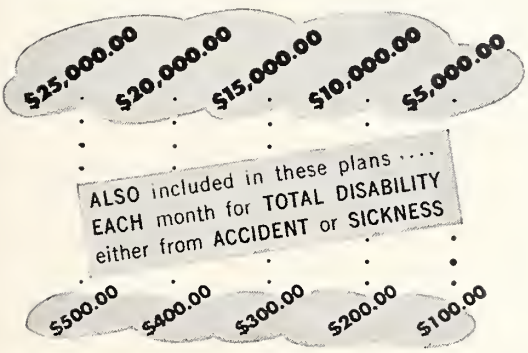
Phone 2-8981 Hutchinson 329 W. 4th St.

Something NEW is Cooking



MORE INSURANCE NOW AVAILABLE

think! HOW THESE AMOUNTS WOULD HELP IN PAYING ESTATE TAXES IN CASE YOU ARE ACCIDENTALLY KILLED . . .



SPECIFIC BENEFITS ALSO FOR LOSS OF SIGHT, LIMB OR LIMBS FROM ACCIDENTAL INJURY

HOSPITAL INSURANCE also for our members and their families

$4,000,000 Assets
$20,000,000 Claims Paid
52 Years Old

Physicians Casualty & Health Ass'ns.
Omaha 2, Nebraska

THE LATTIMORE-FINK LABORATORIES

Topeka — El Dorado
Kansas

J. L. Lattimore, A.B., M.D., Pathologist
A. A. Fink, A.B., M.D., Pathologist
H. C. Ebendorf, M.T., Serologist
A. C. Keith, B.S., Chemist
L. W. Hull, A.B., Bacteriologist
Walter Norris, A.B., Chemist

Anatomical and Clinical Pathology

Containers Furnished Upon Request

Medical and Surgical Supplies for Doctors of Medicine and Hospitals

Munns Medical Supply Co.

512 Kansas Avenue
Topeka, Kansas

facts bluntly, universally must need to know more of the problems and their solutions in the ever-expanding realm of physical disabilities will glean from this book many things of immediately useful value. Medical students, nurses, physical and occupational therapists, and other paramedical individuals will profit from its contents. Finally, the manner in which the material is presented, together with the modest cost of the book, makes it one which the physician can recommend to the lay individual, handicapped or not. Those who have any dealing with the physically handicapped person will immediately recognize the service rendered by Dr. Rusk and his associates in the compilation of the contents of this book.—*D.L.R.*

Textbook of Pediatrics. Edited by Waldo E. Nelson with the collaboration of 70 contributors. Sixth Edition. Published by W. B. Saunders Company, Philadelphia. 1581 pages, illustrated. Price $15.

Students of pediatrics are blessed by having two authoritative, complete, and up-to-date textbooks for study and reference. Publication last year of an excellent new edition of an old favorite, Holt's *Diseases of Infancy and Childhood,* threatened a monopoly enjoyed in recent years by the Mitchell-Nelson textbook. This threat has been met by the new Sixth Edition of Nelson's *Textbook of Pediatrics.*

The new edition is a little shorter than the previous one, yet new material has been added and several sections are treated more completely. This has been accomplished by a much-needed tightening of the entire text. The book is made easy for reference by an index which is exceptional for its completeness and legibility. The large section on growth and development, an outstanding feature of the last edition, has been continued with the many tables of physical measurements. These alone make the book a valuable reference work for any physician concerned with the care of children.—*C.A.M.*

America's pharmaceutical industry is spending approximately $60,000,000 a year for research, reports John A. MacCartney of Parke, Davis and Company, who added that new research laboratories have been built, or are planned, by practically every major pharmaceutical manufacturer.

"The reason is two-fold," he said. "The passing years amply demonstrate the fundamental soundness of a heavily-financed and adequately-staffed research installation. Research tends to be a self-perpetuation effort. Each new discovery opens new avenues of problem approach and new vistas of jobs to be done."

QUESTION?

Have you enrolled in the KANSAS MEDICAL SOCIETY Approved DISABILITY INCOME PLAN?

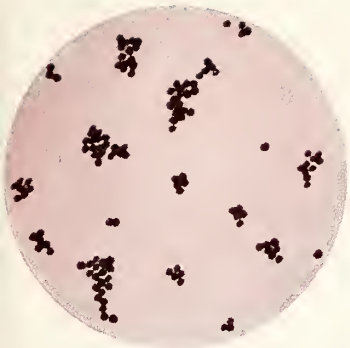
- **Endorsed and Supervised by Your Society**
- **Non-Cancellable Features**
- **Lifetime Accident Benefits**
- **Five-year Sickness Benefits**
- **30% to 50% Saving in Cost**
- **Over 65% of Eligible Membership Now Enrolled**

Officially approved and endorsed by the Council and the House of Delegates.

For Complete Information Write—Kansas Medical Society, 315 West Fourth, Topeka, Kansas

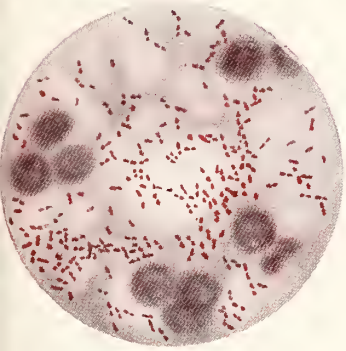
or

FIRST INSURANCE AGENCY
Washington National Insurance Company
639 New England Building Topeka, Kansas



in bacterial endocarditis

"Within ten days [after therapy with CHLOROMYCETIN was begun] there was a dramatic improvement in the patient's clinical appearance and the sedimentation rate and temperature became normal."[4]



in rickettsial diseases

"Chloramphenicol [CHLOROMYCETIN] has been used with striking success in patients with scrub typhus, murine typhus, Rocky Mountain spotted fever, and epidemic typhus."[5]

mycetin®

(Chloramphenicol, Parke-Davis)

CHLOROMYCETIN is a potent therapeutic agent and, because certain blood dyscrasias have been associated with its administration, it should not be used indiscriminately or for minor infections. Furthermore, as with certain other drugs, adequate blood studies should be made when the patient requires prolonged or intermittent therapy.

MEDICAMENTA VERA

PARKE, DAVIS & COMPANY
DETROIT 32, MICHIGAN

TABLE OF CONTENTS

DECEMBER, 1954

Original Articles

The Hypoprothrombinemia-Inducing Activity of Warfarin Sodium (Coumadin Sodium)—Shepard Shapiro, M.D., New York, New York 687

Gallstone Ileus—Edward A. Scollin, M.D., Wichita, Kansas 692

Hypersplenism Associated with Leukemia—Lee Duncan Hanes, M.D., Kansas City, Missouri 697

Arrhenoblastoma—G. E. Kassebaum, M.D., El Dorado, Kansas 703

Editorials

New Year's Resolution 707

Federal Aid in Kansas 707

A Message from the Auxiliary 708

Age and Distribution of Doctors 709

Miscellaneous

President's Page 706

Biopsy Procedures in Office Practice—Tumor Conference 712

Parkinson's Disease—Glen G. Halliday, M.D. —Senior Thesis 721

Entered as second-class matter at the post office at Fulton, Missouri, June 19, 1953, under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of October 3, 1917, authorized on July 2, 1918. Office of Publication: 1201-05 Bluff Street, Fulton, Missouri.

THE JOURNAL OF THE KANSAS MEDICAL SOCIETY

Editorial Board

Orville R. Clark, M.D., *Editor,* Topeka

John W. Cavanaugh, M.D., Topeka

David E. Gray, M.D., Topeka

Richard Greer, M.D., Topeka

Dwight Lawson, M.D., Topeka

Associate Editors

Donald P. Trees, M.D., Wichita

Glen R. Shepherd, M.D., Kansas City

Information

Ownership: The Journal is a non-profit publication owned and published monthly by the Kansas Medical Society.

Subscription: A year's subscription to the Journal is included in membership in the Kansas Medical Society, with $2.00 of each member's dues apportioned to the Journal. Rates to others, except in foreign countries, $4.00 per year or 60c per copy.

Material: Scientific articles, editorials, and data of general interest are invited from all members. Articles are to be submitted on condition that they are contributed solely to this publication. A right is reserved to reject any material deemed unsatisfactory.

Manuscripts: Only manuscripts that are typewritten on one side, double spaced, and original copies can be accepted. Manuscripts will be returned upon request.

Advertising: All advertising contracts, and all copy from advertisers under contract are subject to approval of the editorial board. Copy should be received by the 20th of the month immediately preceding the month of publication.

Non-Responsibility: Although an effort is made to publish only accurate articles and legitimate advertisements, the Journal denies legal responsibility for any statements, opinions, or advertisements appearing under the names of contributors or concerns.

Pauline Farrell, Managing Editor
and Advertising Manager

Oliver E. Ebel, Business Manager

THE KANSAS MEDICAL SOCIETY

President . . . . . Murray C. Eddy, M.D., Hays

Past President . . . . . Lucien R. Pyle, M.D., Topeka

President-Elect . . . . . John M. Porter, M.D., Concordia

First Vice-President . . . Clyde W. Miller, M.D., Wichita

Second Vice-President, Conrad M. Barnes, M.D., Seneca

Constitutional Secretary, James A. Butin, M.D., Chanute

Treasurer . . . . . John L. Lattimore, M.D., Topeka

A.M.A. Delegate, 1954-1955, George F. Gsell, M.D., Wichita

A.M.A. Delegate, 1955-1956, Laurence S. Nelson, M.D., Salina

Councilors and Councilor Districts

First District, Frederick E. Wrightman, M.D., Sabetha

Second District . . . Glenn R. Peters, M.D., Kansas City

Third District . . . . . H. Penfield Jones, M.D., Lawrence

Fourth District . . . . Charles E. Vestle, M.D., Humboldt

Fifth District, Severt A. Anderson, M.D., Clay Center

Sixth District . . . . . Floyd C. Taggart, M.D., Topeka

Seventh District . . . . . To be announced

Eighth District . . . . . James E. Hill, M.D., Arkansas City

Ninth District . . . . Laurence S. Nelson, Jr., M.D., Salina

Tenth District . . . . . Harold M. Glover, M.D., Newton

Eleventh District . . . . Norton L. Francis, M.D., Wichita

Twelfth District . . . . . Cyril V. Black, M.D., Pratt

Thirteenth District . . . . Lloyd W. Reynolds, M.D., Hays

Fourteenth District . . . . Justin A. Blount, M.D., Larned

Fifteenth District . . . . Robert G. Klein, M.D., Dodge City

Sixteenth District . . . . . James L. Jenson, M.D., Colby

Seventeenth District . . . . . H. Preston Palmer, M.D., Scott City

Oliver E. Ebel, Executive Secretary

Rueben M. Dalbec, Executive Assistant

Executive Office: 315 West Fourth Street, Topeka

THE JOURNAL
of the
KANSAS MEDICAL SOCIETY

Copyright, 1954, by The Kansas Medical Society

Volume LV DECEMBER, 1954 No. 12

The Hypoprothrombinemia-Inducing Activity of Warfarin* Sodium (Coumadin® Sodium)

Shepard Shapiro, M.D.
New York, New York

It is possible to prevent or limit thrombosis and thrombo-embolism by rendering the blood hypocoagulable. To achieve this there are available two types of clotting inhibitors: (1) heparin and heparinoid substances which extend the whole blood coagulation and (2) bishydroxycoumarin (Dicumarol®) and similar agents which reduce the level or activity of plasma prothrombin. Heparin was introduced for clinical use in the middle 30's by Swedish and Canadian investigators,[1] and the 4-hydroxycoumarin drug was discovered at the University of Wisconsin by Professor Karl Paul Link and his students in 1939.[2] This paper deals only with 4-hydroxycoumarin compounds.

In the treatment of acute thrombotic disorders the immediate goal is to prevent extension of intravascular clotting and to avoid liberation of emboli from the thrombotic mass. As has been indicated, this can be accomplished by rendering the blood hypocoagulable, and if a 4-hydroxycoumarin drug is used a satisfactory clinical response can usually be achieved by prolonging the prothrombin time as estimated by the single-stage method to the range of "therapeutic" hypoprothrombinemia.

It has been universal experience that certain difficulties have been encountered when Dicumarol® or a related compound has been employed. The hypoprothrombinemia response may be uncertain and irregular in onset, intensity, and duration. These drugs are all administered orally, and variability of absorption from the digestive tract has been a contributory factor. Because of these facts more desirable compounds have been sought.

Several years ago I pointed out that a desirable anticoagulant should possess a high degree of therapeutic activity, be completely and promptly absorbed, exhibit predictable response with early onset of effectiveness and minimum hazard of hemorrhage, and be without other adverse effects.

The purpose of this paper is to describe a new and more dependable drug for the therapy of thrombotic and thromboembolic disorders. It is a 4-hydroxycoumarin compound which possesses the desirable characteristics described above more than any related drug in current use.

After Professor Link and his co-workers isolated Dicumarol®, they screened many analogues for their hypoprothrombinemia-inducing activity. As a by-product of this investigation, one compound, Warfarin (3 (α-acetonyl benzyl)-4 hydroxycoumarin) was recommended by Professor Link for rodent control. It was found that the hypoprothrombinemia observed in various experimental animals with a given dose of Warfarin was generally more reproducible than that induced by Dicumarol® and that it was readily reversed by water-soluble preparations of menadione and by K_1. It was also noted that Warfarin exhibits greater potency and earlier onset of action than

* (3-(α-acetonyl benzyl)-4 hydroxycoumarin)

Presented at the annual meeting, Kansas Medical Society, Topeka, Kansas, May 5, 1954. Dr. Shapiro is assistant professor of clinical medicine, New York University College of Medicine.
Coumadin Sodium® was prepared by Dr. Samuel M. Gordon, Endo Products, Inc., Richmond Hill, New York.

the parent drug. Because of these facts I made clinical trials with the new drug in man.

Initial unpublished observations were dramatically confirmed by an accident reported by Holmes and Love[3] of a patient who took large doses of Warfarin in a suicide attempt on six successive days, greatly in excess of the therapeutic dose now employed safely in man. They confirmed that Warfarin was qualitatively identical with Dicumarol®, that it was highly susceptible to counteraction by menadione, and that it caused no adverse side effects. Only bleeding followed the excessive dosage.

On the basis of these data it was postulated that if Warfarin, which is insoluble, could be prepared in a form suitable for parenteral administration, it would be preferable to any of the drugs now in clinical use. A stable sodium salt of Dicumarol® cannot be made, and the same holds for cyclocumarol which cannot be converted into a sodium salt because of the fact that it has no free hydroxyl group. Warfarin was made available as the sodium salt, and Professor Link supplied me with this material. It possesses the following characteristics:

1. It is soluble in water.
2. It is effective intravenously, intramuscularly, or orally.
3. Relatively small doses are required to induce therapeutic hypoprothrombinemia.
4. Initial prolongation of prothrombin time occurs earlier than after Dicumarol®, cyclocumarol, or ethyl biscoumacetate (Tromexan®).
5. Its tendency to produce hemorrhage from overdosage is less than with the three 4-hydroxycoumarin drugs mentioned above.
6. It yields a smooth extended curve of hypoprothrombinemia in contrast to ethyl biscoumacetate which produces frequent fluctuations in prothrombin time.

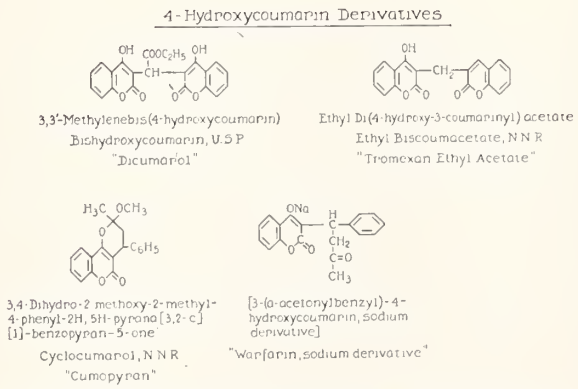


Figure 1. The relationship of Warfarin Sodium to bishydroxycoumarin, ethyl biscoumacetate, and cyclocumarol is illustrated in the above structural formulae. Warfarin Sodium is the sodium salt of the enol form. Stable water-soluble forms of the other 4-hydroxycoumarin anticoagulants illustrated above cannot be prepared.

7. Its effect upon the prothrombin time is readily counteracted by water-soluble menadione or by K_1.

The aqueous solution used by me contains 75 mg. in 2 ml. solution, suitable for intravenous or intramuscular administration. Scored tablets for oral use contain 25 mg. each. Absorption is equally effective by the three routes of administration. I prefer intravenous or intramuscular injection to initiate treatment and during the stages of active thrombosis or thrombo-embolism because it is then of the greatest importance that the blood be rendered and maintained hypocoagulable. After the acute stages have subsided therapy can be continued satisfactorily by oral administration. Therapy can be instituted effectively by mouth also, but for the reasons stated above injection is preferable. Dosages by all routes are at the same level and frequency.

The initial dose is 1 mg. per kilogram of body weight. The smallest starting dose, regardless of weight, is 50 mg., and patients weighing more than 75 kilos are given 100 mg. The new drug, like all other related compounds, must be controlled by reliable and properly spaced estimations of prothrombin time.

I prefer to administer doses by the intermittent method, to be described presently, regardless of the route used. Succeeding doses are given after the prothrombin time extended by the previous dose begins to fall from the highest point on the prothrombin curve. Subsequent doses after the first dose are two-thirds of the initial dose unless the prothrombin time has approximated normal, in which case the same dose first used is repeated. The mode of administration after the first dose is a matter of choice although spaced intravenous injections may be used according to the intermittent dosage schema. Parenteral doses may be given every four or five days, or the drug may be given orally with suitable attention to dose and frequency. If daily maintenance doses are desired, in contrast to the intermittent method, about 12.5 mg. per day or 25 mg. every other day, orally, is usually adequate to retain the prothrombin time of the whole plasma within the accepted therapeutic range of between 1½ and 2½ times normal in seconds, by the single stage method. For intermittent oral dosage I have found 50 mg. every third or fourth day, depending upon the patient's characteristics, usually suffices. Because of the regularity of response after Warfarin, it is usually possible to forecast subsequent doses after the prothrombin time curve for a given individual has been established.

The drug is well tolerated, and no side effects have been noted. Hemograms, liver function tests, and repeated urine analyses have revealed no adverse side reactions. As is true of all anticoagulants,

this drug may induce bleeding from ulcerated or necrotic tissues when the blood is rendered hypocoagulable. Before anticoagulants are administered, the clinician must satisfy himself concerning such risks. In general, however, experience has been that bleeding tendency, such as gross hematuria, has occurred less frequently with Warfarin Sodium than from Dicumarol®, cyclocumarol, and Tromexan®.

Excessive hypoprothrombinemia with or without bleeding induced by Warfarin Sodium is highly susceptible to correction by water-soluble menadione or K_1 emulsion. An added advantage is the fact that K_1 is effective in small dosage and (at least in the rare instances in which I have had to employ it) is not followed by the continuation of normal prothrombin time for many days even in spite of additional doses of anticoagulant. On the contrary, the prothrombin time promptly becomes extended after it recedes to normal. The importance of these phenomena in active thrombotic or thrombo-embolic disease is obvious (Figure 2).

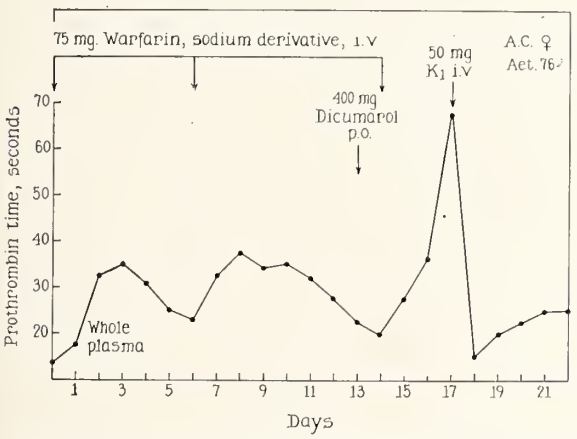


Figure 2. Illustrating excessive prolongation of prothrombin time and response to K1. During recovery phase after second dose, attempt was made to substitute Dicumarol, 400 mg. orally, on the 13th day. Failure or delayed absorption reflected in decrease in prothrombin time on 14th day, and 75 mg. Warfarin intravenously was given. Excessive prolongation to 67 seconds (normal 14 seconds) on 17th day and 50 mg. K1 (Merck) was administered intravenously. Fifteen hours later prothrombin time was 15.5 seconds followed by gradual increase to 17, 22, 25 and 25 seconds respectively on succeeding four days.

In the conduct of anticoagulant therapy the clinician must constantly be alert to the physiologic alterations inherent in the prothrombin producing mechanism. By virtue of this, some patients may be highly susceptible while others may be relatively resistant to the inhibitory agent. It is imperative, therefore, that the therapy be controlled by reliable estimation of prothrombin time. The high order of predictability of response after Warfarin following the establishment of the pattern of prolonged prothrombin time after a given dose and the regularity of this response should place the management of thrombotic and thrombo-embolic disorders with the new drug on a more certain plane than has prevailed to the present.

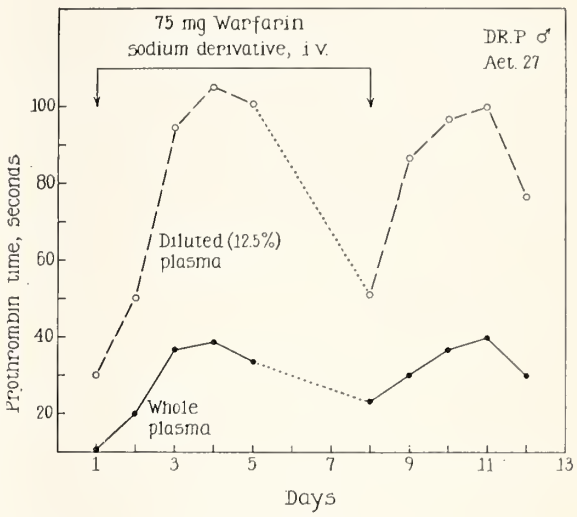


Figure 3. Illustrating intermittent doses and regularity of response after 75 mg. Warfarin Sodium was given on 1st day and 8th day. Note plateau at therapeutic level after each dose lasting three days and selection of points on curve when prothrombin time estimations (6th and 7th days) can be safely omitted.

The therapeutic efficacy of an anticoagulant drug is determined by the facility with which it renders the blood hypocoagulable and maintains the state of reduced coagulability. Since the objective of inducing hypoprothrombinemia is for its favorable influence upon thrombosis and embolism, it is essential not only to induce adequate hypoprothrombinemia initially but also to continue hypocoagulability for the desired period of therapy. The susceptibility to further thrombosis in a patient who already has sustained a thrombotic episode can be overcome by inducing and continuing a state of hypocoagulability.

While the doses given above are recommended on the basis of clinical experience with the drug, it must be borne in mind that each patient responds according to his own individual characteristics. Fortunately the majority of persons react according to a pattern after Warfarin, although there are some persons who exhibit excessive responses while others are relatively resistant. It is because of this that there must always be individualization of dosage determined by the response pattern after the initial doses used. I pointed this out in the first symposium on Dicumarol® held in Atlantic City in 1942. This advice is no less true today than it was at that time. As I see it, if this point of view had been more generally accepted, there would have been less cause for alarm from induced hemorrhage from any of these drugs.

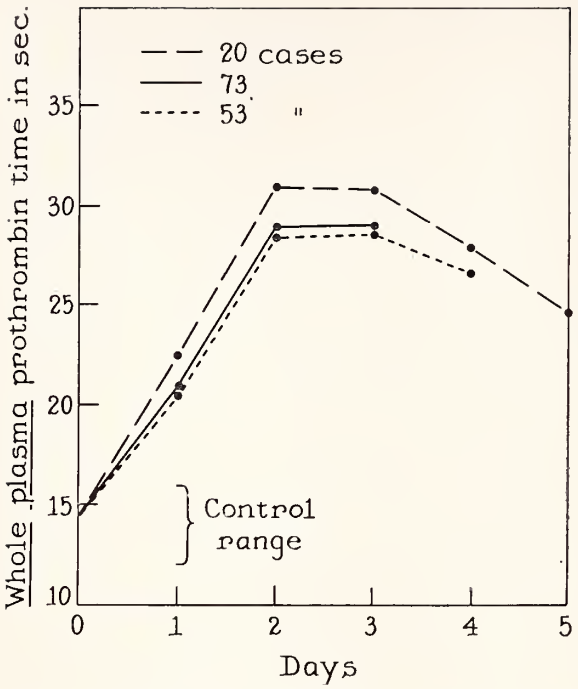


Figure 4. Illustrating mean responses in 20, 53, and total 73 cases respectively. Shows regularity and predictability of response pattern, all 3 curves being statistically practically superimposable.

Generally it is adequate for therapeutic purposes to prolong the prothrombin time of the whole plasma to between 1½ and 2½ times normal in seconds as determined by the Link-Shapiro modification of the Quick one-stage method. A normal of 15 seconds has been reliable in my hands, although a few workers prefer a normal of less than 15 seconds. In any event, constancy and reproducibility of the method employed are essential for the proper adjustment of dosage.

The accepted therapeutic range stated above is not infallible. Why some subjects exhibit rethrombosis when the prothrombin time is within the accepted therapeutic range is a subject for conjecture. When such events occur the clinician must re-evaluate the problem in the framework of the known phenomena, including the technic used for estimating prothrombin time.

It must be borne in mind that intravascular thrombosis represents the culmination of the interplay of a number of factors which need not be identical in every case and in which the state of the coagulability of blood usually represents the common denominator. It is because of this that rendering the blood hypocoagulable favorably influences thrombosis and thrombo-embolism. However, if the activity of other contributory causes becomes overwhelming they might counterbalance the limiting effects of hypocoagulability and, under such conditions, it is conceivable that the therapeutic efficacy of anticoagulants might be inadequate. Outstanding examples of this are cases of coronary artery thrombosis in which the closure of the vessel by dynamic obstructive sclerosis and spasm is so complete that anticoagulation is inadequate to alter the mechanism. Similarly, the thrombotic complications of sickle cell disease crises cannot be altered by rendering the blood non-coagulable.[4]

During the stages of active thrombosis or embolism I prefer to prolong the prothrombin time to the fullest therapeutic extent rather than to err on the side of inadequate dosage. Physiologically this is sound and clinically it is safe, particularly with Warfarin Sodium because of its ready reversibility by Vitamin K. Experience has shown that the incidence of bleeding following initially induced hypoprothrombinemia is relatively low as compared with that occurring when the prothrombin time has been maintained within a constant therapeutic range for an extended period. Why this is so is a subject for conjecture. Just as thrombosis is a multifaceted reaction, so is bleeding after overdosage a reflection of a combination of changes of which hypocoagulability is only one.

Another and essential element is, for want of a

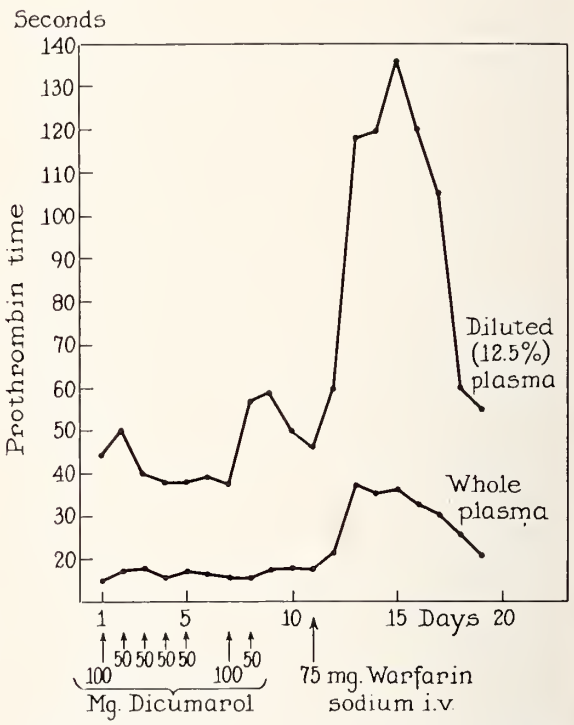


Figure 5. Illustrating the advantage of intravenous administration of Coumadin Sodium versus oral Dicumarol. Inadequate response in the case of a referred patient receiving "maintenance" doses of Dicumarol. On the 7th day pulmonary embolism occurred in spite of additional Dicumarol. On the 11th day, 75 mg. of Coumadin Sodium, given intravenously, induced a satisfactory therapeutic hypoprothrombinemia with cessation of emboli and followed by prompt recovery.

better description, identified as the integrity of the vascular wall. That this is real and how it is influenced can be appreciated from later remarks in this paper. They indicate that continued maintenance of hypocoagulability may impair the vascular wall integrity, which in turn may eventuate in hemorrhage whilst intermittent rise and fall of the level of coagulability permits the vascular wall to remain in a more normal state of integrity with avoidance of bleeding.

The genesis of the intermittent method may be of interest as it relates observations in animals to use in man. Early in 1940, while visiting Professor Link at the University of Wisconsin, I was shown two groups of dogs being treated experimentally with Dicumarol®. One group was given the drug daily; the other group was given equivalent quantities at intermittent intervals. After a while spontaneous hemorrhage occurred in the first group, but no bleeding occurred in the second series of dogs although the prothrombin time was substantially prolonged. These observations in dogs, plus the experience of farmers who found that those cattle ingesting spoiled sweet clover hay every day for many days contracted the hemorrhagic disease but those animals who received feed other than sweet clover hay intermittently did not manifest the hemorrhagic disorder, gave me the clue to the dosage schema which I established when I began to use Dicumarol® in man in 1940. Because of the relationship of these phenomena in dogs and cattle to those in man, I designated this the intermittent method. The essentials of the intermittent method are to give a relatively large initial dose and subsequent smaller doses after the prothrombin time has begun to recede from the peak prolongation of the prothrombin time from the previous dose.

This schema has made it possible to anticoagulate very sick patients for extended periods without serious hemorrhagic manifestations. This is in contrast to the so-called maintenance method in which daily or almost daily doses are given to keep the prothrombin time constantly within a fixed therapeutic range. The incidence of bleeding by this method has been substantially higher than with the intermittent method.

Warfarin Sodium parenterally and orally is particularly well suited for intermittent dosage therapy because its effect is initiated relatively early and continues within the accepted therapeutic range for several days after a single dose of therapeutic magnitude. The characteristics of the drug promise to simplify the management of thrombosis and embolism. This is one of the most serious problems confronting the clinician today since these disorders are most common in older age group patients now occupying the special attention of the profession.

SUMMARY

The foregoing may be summarized as follows:

1. A new hypoprothrombinemia-inducing agent has been described.
2. It can be given orally and, because its sodium derivative is highly soluble in water, its solution is suitable for intravenous or intramuscular administration.
3. Relatively small doses of the drug provide therapeutic hypoprothrombinemia.
4. It becomes effective earlier than the other 4-hydroxycoumarin anticoagulants.
5. It exhibits a low hemorrhage-producing tendency.
6. It causes no adverse side-effects. Only bleeding follows over-dosage, and this is readily corrected by water-soluble menadione and K_1.
7. It exhibits an almost uniformly smooth extended curve of hypoprothrombinemia.
8. It has a high predictability of prothrombin response.

The essentials and advantages of the intermittent method for which Warfarin Sodium is especially suited have been presented.

BIBLIOGRAPHY

1. Jorpes, J. Erik: Heparin in the treatment of thrombosis, Oxford University Press, N. Y. & London 1946.
2. Link, K. P.: The anticoagulant from spoiled sweet clover, Harvey Lecture Series 39:162, 1943.
3. Holmes, R. W., and Love, J.: Suicide attempt with warfarin, J.A.M.A. 148:935 (Mar. 15) 1952.
4. Murphy, R. C., Jr. and Shapiro, S.: The pathology of sickle cell disease, Ann. Int. Med. 23:376, (Sept.) 1945.

Education is not merely the accumulation of facts or information. The process involves the assimilation of knowledge so that the learner becomes a different, a more effective, more useful and happier person than he would otherwise have been.

—*Abbott Kaplan*

Gallstone Ileus

Edward A. Scollin, M.D.

Wichita, Kansas

INTRODUCTION

Gallstone ileus is of the class of rare but too often highly fatal and missed diagnostic entities. Any surgeon or physician coping with acute abdominal cases in the older age patient should consider the probability of obstruction by gallstones in his résumé of pathological possibilities.

Its history is not new; in 1890 Courvoisier reported a series of such cases, and as early as 1654 Bartholin published a report of gallstones perforating into the small intestines. Von Wagoner analyzed a series of 334 cases in 1914, and Moore estimated in 1925 that about 400 cases of gallstone ileus had been reported at that time.

INCIDENCE

Gallstone ileus occurs most often in the older age patient between the seventh and eighth decades. It is most frequent in women giving histories of previous pregnancies and/or chronic digestive disturbances suggestive of biliary pathology or dysfunction.

A statistical incidence of gallstone ileus occurring in all cases of intestinal obstruction has been established, from a number of case study surveys, to range from 0.4 to 5 per cent with a figure of 1 to 2 per cent probably being more accurate.

MORTALITY

From an incidence percentage gallstone ileus would not seem too important, but mortality statistics speak otherwise. A surgical entity with a reported mortality range of 33 to 93 per cent, where such is preventable by early diagnosis and early adequate treatment, becomes a diagnostic possibility always to be remembered when dealing with intestinal obstruction in the elderly patient. Moore in 1924 found that the reported mortality of various authors ranged from 50 to 100 per cent. The present average mortality is still about 50 per cent.

PATHOGENESIS AND PATHOLOGY

Anatomically and pathologically, obstructive gallstones usually gain entrance to the intestinal tract initially by an abnormal communication between the biliary passages and the gut, such most often occurring between the gallbladder and duodenum. Wakefield, Vickers, and Walters[1] in their study of 152 cholecysto-enteric fistulae, representing 140 from the literature from 1922 to 1942 and 10 cases of their own, found a breakdown of 101 into the duodenum, 11 into the jejunum or ileum, 33 into the colon, and 7 into the stomach.

Rigler, Borgman, and Noble[2] feel that cases previously reported in which gallstones causing the intestinal obstruction had apparently passed through the ampulla of Vater had actually passed into the intestinal tract by way of a choledochoduodenal fistula near the end of the common duct. They present two cases in which such findings were seen at autopsy.

The etiological origin of such abnormal biliary intestinal tracts is still a matter of conjecture. Hinchey[3] feels such are resultant of obstruction of the common duct, and paradoxically these cholecysto or cholecystodocho-enteric fistulae are thus "life-saving" incidents. Coffey and Willox[4] are of the opinion that a cholecystitis with cholelithiasis must invariably precede the existence of a biliary intestinal tract fistula, causing a resultant inflammatory fixation of the gallbladder or portion of the biliary tree with eventual erosion into the adjacent bowel.

Fistulae arising from the biliary system do not always connect or empty into the gut. Multiple fistulae have been reported both at surgery and at autopsy and have varied in their communication from the biliary tract to the bronchial tree, renal pelvis, uterus, ovary, bladder, and pleural and pericardial cavities. Open fistulous tracts may not always be demonstrable at surgery, having often become partially or completely fibrosed from the time of original disease to the time of surgery or autopsy.

Fortunately most gallstones entering the gut are subsequently passed or vomited without causing serious obstructive symptoms. Gutman[5] estimates that fewer than one-half of all gallstones that pass into and through the gut cause obstruction.

Obstruction when present need not initially be on a mechanical basis alone. Wangensteen feels such may start as a paralytic or spastic ileus type caused by local irritation from a stone too small to block the gut lumen alone. Subsequently, with spastic reduction of the gut lumen, mechanical obstruction occurs. This mechanism could explain occurrence of intermittent obstruction in these patients; with reduction of spasm, the stone passes on until it becomes impacted again at a more distal site. It has been generally felt, as

Resident in Surgery, Veterans Administration Center, Wichita, Kansas.

Barnard[7] stated, that stones must be 2½ centimeters or more in diameter to cause obstruction. Reduced gut lumen due to adhesions or scarring resulting from regional enteritis, diverticulitis, or other causes tends to obviate this dictum.

Obstructing gallstones have been found most frequently at the ileum. Courvoisier's[8] figures of 65.4 per cent in the ileum, 21.4 per cent in the duodenum or jejunum, 10 per cent at the ileocecal valve, and 2.4 per cent at the sigmoid flexure, are fairly accurate when compared with figures quoted by Foss and Summers,[9] who found out of 150 cases, 59 per cent having stones in the terminal ileum and 3.3 per cent in the colon or rectum. Understandably gallstones can conceivably lodge and have been reported as lodging in the appendix, Meckel's diverticulum, or other intestinal tract diverticula. Multiple stones may be present in the gut at any one time. In general it is felt that those stones entering the colon directly rarely cause obstruction and are usually passed with minimal or no difficulty.

Additionally, intestinal obstruction has been reported by Mayo, Robson, and Smith[10] as being due to volvulus caused by increased weight of the proximal loop from an accumulation of gas and fluid proximal to the gallstone. Rarely, although it has been reported by Wakefield, Vickers, and Walters,[1] obstruction can be caused by intussusception with the fixed gallstone as the point. The same authors report three cases of obstruction due to gallstone without the calculus actually being in the gut. In these cases the stone was lodged in the mesentery of the small gut, and a local abscess reaction caused subsequent development of intestinal obstruction.

Pressure ulceration and necrosis of the obstruction site and a perforation at the point of obstruction or above the point of obstruction, due to excess dilatation of the proximal loop, are pathological possibilities to be expected in the relatively late and often fatal cases.

CLINICAL FEATURES

Diagnosis of gallstone ileus is made by initially considering the possibility of this entity in an older age patient in the sixth or seventh decade presenting the clinical signs and symptoms of obstruction. A history suggestive of chronic dyspepsia or biliary pathology or dysfunction may or may not be elicited. Additionally there may be a history of previous intermittent partial or complete intestinal obstructions with fairly rapid recovery with or without conservative therapy. Dehydration, elevated nonprotein nitrogen, and blood chemistry imbalances are late findings. As with some cases of intestinal obstruction, gas and feces may be passed distal to the obstruction point, and such a finding should not mislead the physician.

X-ray is often diagnostic if the condition is kept in mind. To be looked for are roentgen findings of a partial or complete gut obstruction or ileus accompanied by the presence of gas in the biliary tract. Rigler, Borgman, and Noble[2] report gas seen in the biliary tree in the preoperative films in 13 out of 14 cases of gallstone ileus. They recommend that in consideration of such cases an anteroposterior or posteroanterior film centered over the right upper quadrant is best for visualization of gas or contrast media in the biliary tract. The finding of gas in the biliary passages is seen in only one other condition, an infection of the biliary area with a gas-forming organism.

Failure to visualize gas in the biliary tree even after repeated studies may be explained, as has been previously noted, by the fact that many of these biliary fistulae tend in time to close off by fibrosis. Additionally in the x-ray diagnostic surveys, where gallstones have been seen in previous films, any change in their positions should be sought and noted. Obviously x-rays will often help locate the site of obstruction, although distention of gut loops may be minimal or absent in those cases where obstruction is so often characteristically intermittent.

Right and left lateral decubitus views and supine and upright position x-rays of the abdomen are usually sufficient although, as noted before, close up views of the right upper quadrant area may be additionally helpful. Root[11] points out that pre-x-ray preparation with enemas or laxatives is not advisable; laxatives are omitted for obvious reasons and enemas because confusing air or fluid shadows may be introduced, thus obstructing diagnostic efforts rather than aiding such. Root has suggested using, and has used himself in questionable cases, a thin barium mix either swallowed or passed by way of a Miller-Abbott tube for localizing the points of obstruction. He introduces 20 to 30 cc. of such a mixture and uses both fluoroscopic examination and survey films. By this means, outlining of non-opaque stones should be possible. Barium enemas have been used more often and more acceptably for equivalent purposes.

DIFFERENTIAL DIAGNOSIS

Acute gastrointestinal obstruction from any cause in the older age patient is to be considered in the differential diagnosis when the gallstone ileus patient is first seen. Determination of previous abdominal surgery will aid in ruling in or out the common possibility of obstruction by adhesions. Absence of a history of chronic weight loss and anemia would tend to diminish the possibility of neoplasm. Enterocolitis with its intermittent symptoms may often be confusing, but a febrile response and an elevated white count are more often presented with this condition;

obstruction, when such occurs, is generally progressive and of slow onset.

Essentially a history of chronic biliary or digestive disturbances or a history of intermittent partial or complete intestinal obstruction in the older age patient presenting symptoms of acute intestinal obstruction, where no weight loss or anemia is noted and no previous abdominal surgery has been performed, should suggest the strong possibility of gallstone ileus. X-ray is diagnostic in a good percentage of cases. Conversely, x-ray is not infallible, as witnessed in the case report by Moloney[12] where a preoperative diagnosis, made essentially by x-ray, of gallstone ileus proved at surgery to be a calcified obstructing fecolith in the appendix. In a number of cases, as with other abdominal pathological states, the final diagnosis will be made only at surgery.

TREATMENT

Treatment is almost always surgical. Wangensteen[6] initially advocated, on occasion, the use of suction and antispasmodics in the hope that the stone would pass spontaneously. In the patient who will not tolerate surgery, this may be indicated as a last resort, but of late years the feeling is that suction should be an adjunctive preoperative or postoperative measure only. Used as a completely therapeutic measure, suction can confuse the diagnosis, mask any subsequent perforation, and lull the patient and physician into a sense of false security.

The second case reported here illustrates this point quite well. General supportive measures such as intravenous fluids, electrolytic replacement, etc., are obviously indicated as required.

Operative excision of the obstructing stone with or without resection as required or indicated should be carried out as soon as the patient can be prepared to tolerate surgery. The initial incision should be over the point of obstruction if such is known, otherwise a right rectus approach can be used and extended as required. "Milking" the stone distally if possible or proximal, if proximal gut dilatation is not excessive, has been considered preferable since a more physiologic bowel wall for repair is secured. Coffey and Willox[4] prefer milking the stone proximally about one foot from the site of obstruction and removing the impacted stone through a small longitudinal incision after isolating the bowel segment with rubber shod clamps. The incision is later closed transversely. Attempts to crush the stone at surgery and trust to later "spontaneous passage" are to be definitely condemned.

Enterostomy proximal to the point of obstruction is rarely indicated now if a Cantor or similar type tube can be successfully passed. Where the obstructing stone is in the large bowel, especially the sigmoid, removal of stones with immediate bowel wall closure or resection with primary anastomosis may be unwise and unsafe. In such cases a proximal or a transverse colostomy, where feasible, can be performed, and operative removal of the stone with or without resection can be carried out at a later date when the patient represents a better surgical risk.

A search for one or more additional stones in the intestinal tract should be made; if more are found, they should be removed to prevent possible subsequent obstruction. The second case serves as an example of this point. X-ray or palpable evidence of gallstones in the gallbladder at surgery is an obvious indication for later cholecystectomy procedure assuming adequate biliary drainage. Essentially then, the main purpose surgically is to relieve obstruction the fastest and safest way possible. Accordingly, cholecystectomy, reparative surgery for present fistulae, etc., are secondary procedures to be done as needed at a later date.

The question of subsequent operative closure of biliary intestinal fistulae is a more difficult problem. As previously noted, Hinchey[3] feels that these fistulae result from obstruction of the common duct and paradoxically may be "life-saving" occurrences. There would seem to be a good deal of truth in this conjecture; in a number of patients operated upon for gallstone ileus, the cholecysto or choledocho-enteric fistulous tracts were noted to have become obliterated by fibrosis following apparent re-establishment of adequate biliary drainage through the usual physiological channels. A physiological approach, when stones are present in the gallbladder, would appear to be a cholecystectomy if adequate drainage of bile is present or can be obtained by surgical anastomosis. (When a choledocho-enteric fistula concurrently or separately exists, the same reasoning would apply plus the additional surgical procedure of closing such a fistulous tract.)

Follow-up cholecystectomy has been proposed as a prophylactic surgical procedure in the older patient with cholelithiasis, with or without a previous history of partial or complete intestinal obstruction by gallstones. This holds for cases where the patient may not be having or have had symptoms from the cholelithiasis.

Concerning cholecysto or choledocho-colonic fistulae, there is a difference of opinion as to further surgery. Wakefield, Vickers, and Walters[1] are of the belief that there is a frequent occurrence of cholangitis and hepatitis, diarrhea, and weight loss in such patients, and accordingly such fistulae should be closed at the earliest possible time. Other authors do not agree. It would seem advisable to close such fistula surgically and do an associated cholecystectomy where cholelithiasis is present, if, as mentioned be-

fore, adequate biliary drainage by the normal channels is present or can be secured by anastomotic surgical procedure.

CASES

The following are the only coded cases of gallstone ileus at the Wesley Hospital, Wichita, from 1920 to January 1, 1952. I wish to express my appreciation to Dr. L. E. Vin Zant and the late Dr. James S. Hibbard of Wichita for permission to present these cases.

Case Number 1: Mrs. C. G., a 62-year-old housewife, entered Wichita Hospital on November 19, 1951, complaining of intermittent colicky abdominal pain of sudden onset for the past four days. Generalized abdominal bloating occurred and was followed by nausea and vomiting of bile stained fluid and undigested food particles. Previous attacks of intermittent abdominal distention and colic, subsequently relieved by bowel action, had been noted off and on since the patient was 40 years old. Severe heartburn, that is substernal and precordial distress, had been noted for the past month.

This patient stated she had experienced indigestion after eating beans, cabbage, onions, and fatty foods. No past history of jaundice or hematemesis was obtained. The patient is a grava V and para V. Emergency scout film of the abdomen on admission showed an area of calcification in the left lower quadrant and small intestinal distention. The patient stated she had had no bowel movement since the onset of her illness and had passed gas only with the aid of enemas.

Past history and systemic review were non-contributory other than as noted.

On the fifth day of hospitalization, she was transferred to Wesley Hospital. Physical examination at that time revealed a well developed, markedly obese, white female in no apparent acute or chronic distress. Temperature was 98.4, pulse 80, and blood pressure 100/70. The patient wore a hearing aid. The sclerae were clear. There was a mild systolic grade I murmur over the aortic area. The abdomen was noted to be markedly obese. Peristalsis was audible. Deep and rebound tenderness was elicited in the right upper quadrant. Laboratory work on admission revealed a complete blood count and urine within normal limits. Serology was negative.

Course in hospital: A diagnosis of gallstone ileus was made. General supportive therapy was immediately started and a Levine tube was passed and connected to continuous suction. The patient was taken to surgery the following morning and a right rectus incision was made. On exploration of the abdomen the terminal ileum was observed to be obstructed by a firm, round, slightly movable mass. The small gut was distended proximally. The obstructing mass was milked distally and the area packed off. The ileum was incised over the mass. A large gallstone measuring 4 x 3 x 3 cm. was removed. The bowel wall was inspected; no areas of necrosis or erosion were seen, and the ileum was closed. The abdomen was



Figure 1. Case 1. Admission scout film of the abdomen showing small intestinal distention with an area of calcification in the left lower quadrant of the abdomen.

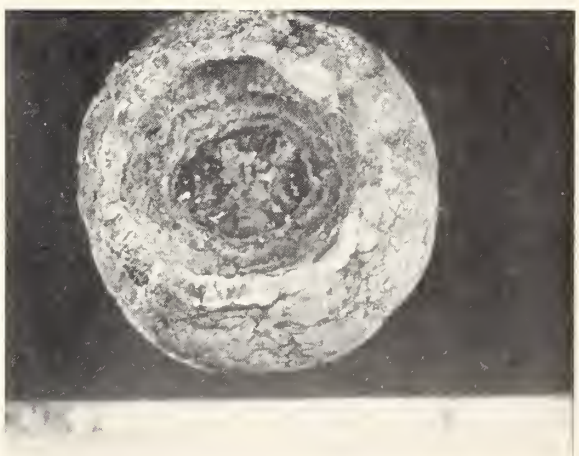


Figure 2. Case 1. The obstructing gallstone removed at surgery measured 4 x 3 x 3 cm.

closed without drainage. The postoperative course was essentially uneventful. Discharge was on the 10th postoperative day.

On January 9, 1952, surgical procedures performed included exploration of the common duct, a cholecystectomy, and closure of a large fistula extending between the gallbladder and the duodenum.

Case Number 2: This case represents two separate admissions and operations and is summarily noted here since it was fully reported previously by Vin Zant and Hibbard.[13]

This 85-year old female presented symptoms of obstruction of four days' duration. X-rays showed a calcified shadow in the right upper quadrant and another calcified shadow in the left midabdominal area at the site of obstruction. A diagnosis of gallstone ileus was made. Conservative therapy because of family wishes was tried for 10 days without success. Surgery was then performed successfully.

About five weeks later the patient presented symptoms of partial intestinal obstruction. X-rays showed an opacity in the left lower quadrant; no opaque shadow was seen in the right upper quadrant although one was so located previously on films five weeks previously. A diagnosis of gallstone ileus was made. Surgery was performed successfully.

SUMMARY

1. The syndrome of gallstone ileus is discussed. Although a relatively rare entity, its usual high mortality is often the result of late or missed diagnosis and delayed adequate surgical treatment. Thus it is an important consideration in the differential diagnosis in an older patient presenting symptoms of acute, partial or complete intestinal obstruction. Clinical features are presented.

2. Treatment of gallstone ileus is essentially and primarily surgical.

3. Follow-up management of patients with reference to any existing cholelithiasis or cholecysto or choledocho-entric or colonic fistulae is still a matter of varying opinion, although subsequent surgical closure of the fistulae or cholecystectomy seems indicated where adequate biliary drainage by natural channels or by anastomotic means is present or can be secured.

4. Two case histories are presented. In each case the diagnosis was made pre-operatively.

BIBLIOGRAPHY

1. Wakefield, E. G.; Vickers, P. M., and Walters, W.: Intestinal obstruction caused by gallstones, Journal of Surgery 5:670-673, 1935.

2. Rigler, L. G.; Borgman, C. N., and Noble, J. F.: Gallstone obstruction, pathogenesis and roentgen manifestations, JAMA 117:1753, 1941.

3. Hinchey, P. R.: Gallstone ileus, Archives of Surgery 46:9-26, 1943.

4. Coffey, R. J., and Willox, G. D.: Gallstone obstruction of the intestinal tract, American Surgeon 18:286-296, 1952.

5. Gutmann, J. H.: Ileus due to migrating gallstones, American Journal of Surgery 30:548, 1935.

6. Wangensteen, O. W.: Intestinal obstruction, Thomas, 2nd Edition, 1947, pages 316-322.

7. Barnard, H. L.: Intestinal obstruction due to gallstones, American Journal of Surgery 36:161, 1902.

8. Wangensteen, O. H.: Intestinal obstruction, Thomas, 2nd Edition, 1947, Pages 316-322—Courvoisier's figures.

9. Foss, H. L., and Summers, J. D.: Intestinal obstruction from gallstones, Annals of Surgery 115:721-735, 1942.

10. Mayo, Robson, and Smith: As reported by Wangensteen in his book *Intestinal Obstruction,* 2nd Edition, 1947, pages 316-322.

11. Root, J. C.: Intestinal obstruction, gallstone impaction, Cleveland Clinical Quarterly 16:154-157, 1949.

12. Moloney, G. E.: Acute appendicitis simulating gallstone ileus, British Journal of Surgery 35:212-213, 1947.

13. Vin Zant, L., and Hibbard, J.: Recurrent ileus due to gallstones, Journal of Kansas Medical Society 50:17-19, 1949.

14. Hand, B. H., and Gilmore, W. E.: Gallstone ileus, American Journal of Surgery 59:72-78, 1943.

15. Williamson, J. C. F. Lloyd: Obstruction of the colon by gallstones, British Journal of Surgery 39:339-341, 1952.

16. Nemir, P. Jr.: Gallstone ileus, report of eight cases, Surgery, Gyn. and Obst. 94:469-474, 1952.

From the beginning of our history, we have been diversified, culturally and racially. It has been a great human experiment which has been launched and conducted on our American shores and plains—perhaps the greatest and most challenging in the history of human society. . . . We have set out here to demonstrate that peoples of all racial strains, of all colors, creeds and cultures, of widely varying background, can be accepted as equals, can learn to live and work and play together and become a firm unity by the force and attraction of noble ideals mutually shared—the dignity and worth of the individual, the equality of all men and the conception of the state as the democratic expression and the servant of free men and women.

—*Ralph J. Bunche*

Hypersplenism Associated with Leukemia

Lee Duncan Hanes, M.D.

Kansas City, Missouri

One of the hematologic complications in patients with leukemia and leukosarcoma which has received increasing attention over the past several years is hypersplenism. The spleen normally exerts control over the number of circulating blood cells by certain inhibitory or destructive mechanisms. Anatomic enlargement of the spleen, whatever the etiology, often results in a physiologic hyperfunction.

Secondary hypersplenism may manifest itself in the form of a hemolytic anemia, produce selective cytopenias such as neutropenia, anemia, thrombocytopenia, or any combination including pancytopenia, which is a reduction in all the cellular elements of the blood, and may be the mechanism in "aleukemic" phases sometimes seen in leukemia. The bone marrow shows hyperplasia for the element deficient in the peripheral blood.

Little is known of the etiologic stimulus except the constant existence of splenomegaly. The exact mechanism is controversial and has been the subject of much investigation.

Doan[13] and his co-workers have for years leaned toward the theory of hypersequestration by the spleen of the deficient blood element or elements, while Dameshek[4] and his associates have championed a humoral hypothesis with the spleen elaborating a substance which inhibits the proper maturation or release from the marrow of the deficient blood elements.

The syndrome which presents the greatest clinical problem and that which has been most extensively investigated is acquired hemolytic anemia related to splenic hyperfunction.

ETIOLOGY AND PATHOGENESIS

In secondary acquired hemolytic anemia, though the immunologic mechanism is obscure, all the classic actions of cell antibodies in vitro have been demonstrated.

Boorman et al.[5] first demonstrated that the immune serum prepared by Coombs, Mourant, and Race[6] by the injection of human serum into rabbits could be used to distinguish certain types of acquired hemolytic jaundice from congenital hemolytic jaundice. They presumed that the agglutination of red cells which occurred was a physical manifestation of the union of the globulin or other substance on the surface of the red cell with the antibody in the test serum. In most cases of immunohemolytic disease the Coombs test is positive, indicating the presence of "coating" antibodies.[25]

In many cases the serum contains free incomplete agglutinins of the warm or cold type,[21] sometimes active in saline but more frequently active in albumin[7] or against trypsin treated red cells.[8] Trypsinizing red cells involves destruction of the surface antigens of the cell with consequent uncovering of deeper structures with antigenic properties. Dodd[9] believes that similarily injured red cells as modified cell antigens may play a large part in hemolytic anemia by initiating auto immunization with production of antibodies with duel specificity for normal and abnormal cells.

A hemolysin is demonstrable in a few cases of acquired hemolytic anemia.[10] Methods have been described by which red cell antibodies can be demonstrated to render cells susceptible to phagocytosis, the opsonic effect.[11]

Administration of small quantities of hemolytic serum in a single injection is sufficient to cause a progressive, fatal hemolytic anemia in the guinea pig with the formation of incomplete auto antibodies, which can be demonstrated in vivo as agglutinins and which possess hemolysing properties.[12]

The actual destruction of these "sensitized" red cells probably takes place in several ways. Intravascular hemolysis is apparently one of the mechanisms, particularly when free auto or isohemolysins are present in the serum. It has been noted in experimental hemolytic anemia in animals that antibodies destroy more red cells in vivo than in vitro.[10] After injection of anti red cell serum into an animal a biphasic reaction occurs. The first phase is one of rapid hemolysis, probably caused directly by amboceptor and complement in the blood. This is followed by a prolonged hemolysis which goes on for several days, generally destroying a larger number of cells and caused by other means, probably a slow mechanical destruction of sensitized cells.[13]

Abnormal red cell antibodies are usually demonstrable as agglutinins. Observations in experimental hemolytic anemia[14] and from direct observation of circulating blood in patients indicates that intravascular agglutination of red cells very probably takes place in immunohemolytic disorders in man. Ag-

The author was graduated from the University of Kansas School of Medicine in 1954 and is now interning at St. Luke's Hospital, Kansas City, Missouri.

glutinated red cells are more susceptible to trauma, and, according to Castle, Ham, and Shen,[15] the agglutination also brings about hemolysis via erythrostasis with release of injurious substances from autolyzing tissues adjacent to stagnating red cells, blocking small blood vessels.

Hemolysis resulting from the more frequently found "coating" antibodies can best be explained on the basis that the red cells are in some way damaged and thus rendered more susceptible to lysis and phagocytosis. Erythrophagocytosis has been described in acquired hemolytic anemia in the spleen and other tissues.[11] The ability of the splenic macrophages to ingest the sensitized or antibody coated cells but not the normal ones was demonstrated by Brandt et al.[16] Erythrophagocytosis cannot explain the spherocytosis seen in vivo but not in vitro, which may be explained by the sequestering of coated red cells in the spleen and other organs.[17] Neither can the hemoglobinemia frequently found in immunohemolytic disorders be explained by phagocytosis alone.

Although the spleen plays an important part in these syndromes, it is obvious from the results of splenectomy that the role of the spleen varies from case to case. Effects range from complete cure to no change. The antibody titer may decrease rapidly or remain high, even though splenectomy has been of good clinical effect.[18] It seems evident that in at least some cases abnormal antibodies are produced by the spleen.

Wright et al.[7] found higher antibody titers in the splenic blood than in the peripheral blood. Wagley et al.[19] incubated normal red cells with spleen tissue from patients with acquired hemolytic anemia and found the incubated cells to be heavily coated with an agglutinating substance. In other cases, particularly where splenectomy causes clinical improvements but no fall in antibody titer, it would appear that red cells altered by antibodies produced elsewhere are retained in the spleen and destroyed in some manner.[17] Failure of splenectomy may be attributed to the fact that other parts of the reticuloendothelial system may be involved in these processes.[15]

CLINICAL PICTURE AND DIAGNOSIS

In hemolytic disease there is increased destruction of red blood cells in the peripheral blood. Anemia occurs when the rate of destruction exceeds the compensatory capacity of the marrow to replace the cells as they are being destroyed. Normally when the red cell has reached the end of its life span it is broken down in the reticuloendothelial system, and particularly the spleen, into a hemoglobin and a pigment portion. The iron and protein portions of hemoglobin are retained within the body while the pigment portion is freed into the blood stream as the normal serum bilirubin. The bilirubin passes via the liver into the gastrointestinal tract where it is reduced and excreted as fecal urobilinogen. A portion of the latter is reabsorbed into the blood stream and reexcreted via the kidney as the normal urinary urobilinogen.

In hemolytic anemia more red cells are destroyed, resulting in a greater liberation of bile pigment into the blood stream, an elevated serum bilirubin, and possibly a jaundiced patient. More pigment is excreted via the liver into the gastrointestinal tract, the excretion resulting in an increased fecal urobilinogen, and concomitantly more of this is reabsorbed into the blood stream to be excreted via the kidneys as an increased urinary urobilinogen.

With excessive hemolysis, therefore, we find evidence of increased hyperplasia involving the erythropoietic elements in the bone marrow, liberation of red blood cells into the peripheral blood with reticulocyte counts as high as 50 per cent, and breakdown of the normal marrow barriers so that younger nucleated red cells may appear in the peripheral blood.

On the other hand, we expect to find evidences of increased blood destruction with elevated indirect plasma bilirubin, increased fecal and urinary urobilinogen, spherocytosis with increased fragility of the red cell, and anemia. The serum bilirubin may be normal, but fecal urobilinogen is always elevated.

An important characteristic of hemolytic anemia is the rapid destruction of transfused red cells in the patient's circulation. Loutit and Mollison,[20] employing the Ashby technique, noted that normal red cells became Coombs' positive and disappeared in 10 to 40 days from the recipient's circulation in contrast to a normal life span of 120 days, which has been substantiated by studying cells tagged with radioactive chromate.[22] The need for large amounts of blood to maintain hemoglobin and red cell count without obvious blood loss points to a hemolytic process.

In acquired hemolytic anemia the most common immunologic finding is a positive antihuman globulin (Coombs') test, though it may be negative[39] and has on occasion been found positive in certain congenital hemolytic syndromes.[7] The indirect Coombs' reaction may be positive when other methods have failed. The serum of these patients usually contains a panagglutinin capable of reacting with cells of their own blood group or normal human group 0 cells, which can be demonstrated by testing in several media at various temperatures.

Erythrocytes treated with trypsin or papain[8] may be used to detect abnormal antibodies. Failure to detect antibodies may be due in some cases to the presence of large amounts of cold precipitable cryoglobulin.[23] Another manifestation of a heightened

capacity to form antibodies is the frequent occurence of biologically false positive tests for syphilis. Heterophile antibody is sometimes produced.[24]

A factor of some diagnostic and therapeutic import is the difficulty often encountered in correctly typing the patient's blood. Because of the marked tendency to auto agglutination, the patient may be incorrectly typed as AB Rh positive. If any suspicion exists, the red cells should be thoroughly washed with saline and typed at 37°C.[32]

THERAPY

1. Transfusions: In hemolytic anemia the effect of transfusion is short and the added difficulty of precipitating hemolytic crises is introduced, which will result in a lower rather than increased red count.[40] In many cases even frequent transfusions are inadequate. Transfusions are primarily useful when given in conjunction with steroid therapy or splenectomy.

2. ACTH and Cortisone: Since these syndromes are apparently due to adverse antibody reaction, the trial of corticotropin and cortisone, known to exert a beneficial effect on diseases of hypersensitive causation, was rational. Dameshek[34] and Gardner[35] were the first to report favorable results in treatment of acquired hemolytic anemia with ACTH. These hormones may decrease the severity of blood destruction, with a rise in hemoglobin, erythrocyte count, and hematocrit and a decrease in serum bilirubin and fecal urobilinogen levels. A positive Coombs' test may or may not become negative.[24] More often there is a marked drop in titer of hemolysins and iso-agglutinins.[24, 37] In some instances the disease is arrested or can be kept under control for months.[39] However, in most cases, remissions can be maintained only with continued therapy.[43, 44]

Dameshek postulated that ACTH may alter the milieu of the red cell, to suppress antibody production or adsorption of hemolytic proteins on the cell surface and thus prevent hemolysis. However, with hemolytic anemia caused with phenylhydrazine, there is no question of immune bodies or adsorption of proteins; yet ACTH and cortisone significantly alleviate anemia caused by this chemical in adrenalectomized rats.[36] Bethel et al.[41] suggest that they modify splenic dysfunction. Gardner[35] found a decrease of mechanical and osmotic red cell fragility in some instances after administration of ACTH. Garcia[38] believes that ACTH primarily increases production and delivery of red cells to peripheral blood.

Cortisone seems to be as effective as ACTH.[39] Most patients treated with transfusions when needed and cortisone, 100 to 200 mg. daily, show some improvement in 7 to 14 days,[32] but the degree and length of response is unpredictable, and continued steroid therapy is usually necessary to maintain results.[42] Smaller doses may be effective for maintenance. A restricted intake of sodium, the addition of potassium salts, and the administration of testosterone to offset negative nitrogen balance must be considered in long term steroid therapy.

3. Splenectomy: Splenectomy in leukemia and leukosarcoma has been reevaluated in the past 10 to 15 years. Until 1940 results were poor and mortality rate high, since it was frequently performed empirically as treatment of the fundamental malignant process which is unaltered by splenectomy. Bryant (1866) was the first to report removing the spleen for leukemia. Billroth (1866) and Kocher (1888) were the first to perform splenectomy for leukosarcoma of the spleen. Mayo, reporting 60 cases of splenectomy in leukemia and leukosarcoma (1928), considered splenectomy to be palliative and indicated in young patients.

A more rational approach was initiated by Ferrata and Fieschi[47] in 1939, recognizing the clinical and hematologic manifestations of hypersplenism as the indication for splenectomy. Since 1940 many reports of small series of cases have been published, indicating that the operation is of value when performed for relief of a co-existing hypersplenism.[26, 33, 39, 40] Good results have been obtained in the majority of cases where presence of hemolytic anemia and hypersplenism, thrombocytopenia or pancytopenia was the criterion. Only in cases in which great discomfort results from an extremely large spleen is splenomegaly without hypersplenism an indication for splenectomy.

The largest series of cases was reported by Fischer, Welch, and Dameshek[33] in March 1952. Splenectomy was performed on 18 patients with leukemia and leukosarcoma, using these criteria. In 8 patients the hemolytic anemia or cytopenic disturbance was controlled from 1 to 5 years. Three received benefit for 3 months. Three patients received no benefit.

For best results, the bone marrow should be hyperplastic, and the finding of a markedly diminished number of megakaryocytes or nucleated red cells in the bone marrow represents a contraindication to splenectomy.[33, 46]

Splenic puncture may be helpful in some cases to rule out extra-medullary hematopoiesis. A trial with ACTH or cortisone before splenectomy is indicated.[44]

Results of splenectomy are not predictable, but in the majority of cases remissions are produced with a mortality rate from operation only slightly higher than that for splenectomy in more favorable situations, in spite of the serious condition of many of these patients. Since management is difficult and prognosis poor in many cases without splenectomy, the operation is definitely a useful measure and probably should be done more frequently in selected cases.

CASE NO. 1

UNIVERSITY OF KANSAS MEDICAL CENTER

An 80-year-old housewife was first seen on December 27, 1953. She complained of progressive weakness and easy fatigability for 2 months following intestinal flu. General health had been good except for bronchial asthma of 30 to 40 years' duration. She had had no attacks during the preceding two or three years. Two years before admission she had an attack of arthritis involving knees and ankles, completely relieved with cortisone. She had bruised easily all of her life. Various hematinics had been tried without improvement. Her skin was pale, and there were numerous ecchymoses on both hands and forearms. There was no lymphadenopathy. Sclerae were slightly icteric, and fundi showed grade 3 arteriosclerotic changes. The heart was somewhat enlarged, and there was a grade 2 aortic systolic murmur and a grade 3 apical harsh systolic murmur. Abdomen was obese, and the tip of the spleen could be palpated far laterally.

Laboratory work on admission gave the following results. Urine showed a trace of albumen. Blood chemistry and electrolytes were within normal limits, as were findings on the kidney, ureter, and bladder examination. Electrocardiograms showed left axis deviation. A chest x-ray revealed an enlarged heart with suggestive evidence of substernal thyroid. Sedimentation rate was 45 in 35 minutes. Gastric analysis was normal. A hemogram showed a red cell count of 1,680,000 with marked anisocytosis and macrocytosis, an occasional spherocyte, and 1 nucleated red cell per 100 white blood count. Hemoglobin was 5.9 grams or 37 per cent, hematocrit 46 per cent, reticulocytes 48.4 per cent with 242,000 platelets, white blood count 27,150 with 77 per cent lymphocytes, 22 per cent polymorphonuclear leucocytes, and 1 per cent monocytes. Indirect serum bilirubin was 2.6 mg. per cent, direct 0.4 mg. per cent. Urinary urobilinogen was 2.6 mg., fecal urobilinogen 348 mg. Prothrombin time was 75 per cent of normal. Red cell fragility test was slightly increased. Direct Coombs' reaction was 4 plus.

Bone marrow study showed erythroid hyperplasia and lymphocytic infiltration with cells of chronic lymphatic leukemia. Megakaryocytes were observed scattered throughout.

A diagnosis of chronic lymphatic leukemia and hemolytic anemia was made.

The patient was given three blood transfusions and then was started on cortisone, 100 mg. daily. Six days later, following two more transfusions, her red count was 3,550,000 with hemoglobin 11.8 grams (76 per cent), reticulocytes 21.4 per cent. The Coombs' test remained 4 plus, serum bilirubin decreased to 1.4 indirect and 0.2 mg. direct, urinary urobilinogen fell to 0.7 mg., and hemoglobin was sustained at 11.8 grams at the end of the one week. However, fecal urobilinogen was 728 mg.

This patient was obviously icteric, with increased serum bilirubin and red cells hemolyzing at a terrific rate, as indicated by the elevated reticulocyte count, and high urinary and fecal urobilinogen, yet she managed to maintain her count fairly well, presumably due to intensive compensatory hyperplasia. Though the reticulocyte count, serum and urinary urobilinogen steadily decreased, fecal urobilinogen remained elevated and the Coombs' test continued strongly positive in spite of cortisone therapy. She was discharged on 100 mg. of cortisone daily.

Cortisone was decreased to 75 mg. per day on February 15, 1954, and on February 22 she was given 3.5 mc. of P_{32} orally. She continued to feel well, but the Coombs' test remained 4 plus, reticulocyte count ranged from 18 to 35 per cent, and hemoglobin gradually dropped to 9.3 grams (60 per cent) with a red count of 2,650,000.

She was admitted to the hospital on April 1, 1954, for transfusions and evaluation. Reticulocyte count was 22.8 per cent, nonprotein nitrogen 45, serum bilirubin indirect 1.5, fecal urobilinogen 453 mg., and urinary urobilinogen 5.7 mg. Because of her advanced age and arteriosclerotic heart disease with calcific-aortic stenosis, constituting a surgical risk, the advisability of splenectomy was debated. It was concluded that the hemolytic process could be controlled for some time with cortisone and transfusions. A blood transfusion of 1,000 cc. was given, and cortisone was increased to 100 mg. daily.

On April 12, 1954, her hemoglobin was 10.8 grams (70 per cent), red count 3,140,000, reticulocytes 16.6 per cent, Coombs' test 4 plus. It was felt that splenectomy was not indicated in view of the relative contraindications to surgery and fairly good response to treatment.

CASE NO. 2

VA HOSPITAL, KANSAS CITY, MISSOURI

A 58-year-old man was first found to have chronic lymphatic leukemia in February of 1947. He complained of weakness and fatigue of one year's duration. His leukemia was adequately controlled and normal red count was maintained with intermittent courses of x-ray until August of 1952, when his red count began to drop. On November 14, 1952, his red count was 1,720,000, hemoglobin 5.5 grams (34 per cent), hematocrit 19 per cent, platelets 112,000, white blood count 18,300 with 62 per cent lymphocytes, 1 per cent monocytes, and 1 per cent eosinophiles.

He had become increasingly weak. Physical examination showed generalized lymphadenopathy. There was an occasional rale in the right lung base. The liver was palpable one finger below the right costal margin. The spleen extended 4 or 5 fingers below the left costal margin and was not tender. A right direct inguinal hernia and bilateral hydrocele were present.

Urinalysis showed 10 to 12 white blood cells per high power field. Bleeding time, clotting time, and red cell fragility were normal. Serum bilirubin indirect was 1.5 mg., direct, 0. Quantitative serology was 4 Kahn units with a negative complement fixation. X-rays of the chest showed infiltration of the right apex and an elongated tortuous aorta with calcifications in the knob. Bone marrow studies showed hyperplasia of the erythroid series with mild lymphocytic infiltration.

On December 5, 1952, after seven transfusions, red count was 3,050,000, hemoglobin 10 grams. He was discharged on hematinics.

The patient was readmitted on December 24, 1952, because of increasing anemia, weakness, and anorexia. Physical findings were essentially the same as before except that the spleen at this time was below the umbilicus and there was 2 plus pedal edema.

Laboratory studies showed a red count of 2,400,000, hemoglobin 7.5 grams, hematocrit 23 per cent, white blood count 7,800 with lymphocytes 56 per cent, indirect bilirubin 1.9 mg. per cent. Secondary hypersplenism with hemolytic anemia was suspected although the Coombs' test was negative.

He was started on cortisone, 50 mg. every 6 hours, and was given 14 transfusions over 36 days. Red count rose to 5,260,000, hemoglobin 14.5 grams, white blood count 27,800 with 77 per cent lymphocytes. Appetite and strength improved. Cortisone dosage was gradually decreased to 25 mg. daily with maintenance of red count. He was discharged January 31, 1953.

He was again admitted on March 7, 1953, with an upper respiratory infection and temperature of 102 degrees. In addition to the previous physical findings, there was a pale icteric tint to the sclerae and the skin.

Red count on admission was 1,320,000, hemoglobin 4 grams, platelets 74,000, white blood count 15,000 with 76 per cent lymphocytes. Serum bilirubin was elevated to 3.1 mg. per cent, all indirect. Urinary urobilinogen was 3 Ehrlich units, and prothrombin time was 45 per cent of normal. Bone marrow showed a definite increase in lymphocytic infiltration but an adequate number of erythroid and myeloid elements. Six transfusions were given.

The febrile illness responded to antibiotics. On March 14, with the red count 3,800,000, hemoglobin 9.6 grams (62 per cent), cortisone dosage was increased to 200 mg. daily. Cortisone was slowly reduced to 100 mg. daily by April 3, with sustained red cell counts at 4,000,000, hemoglobin 10.6 grams. One transfusion was given on April 17, with increase in red count to 4,350,000, hemoglobin 10.8 grams. Cortisone dosage was reduced to 50 mg. daily with a slow steady drop in red cells to 3,200,000 by May 19. On June 1, red count was 2,100,000, in spite of the fact that 3,500 cc. of blood had been given over this period. Cortisone dosage was then increased to 200 mg. daily (June 6), reduced to 150 mg. daily on June 14, and to 100 mg. daily on June 25. A total of 5,000 cc. of blood was given from June 3 to June 28 with no remission in the hemolytic process.

Since control of the hemolytic anemia with cortisone and transfusions was a staggering problem, splenectomy was planned in the hope that some reduction in the number of transfusions and amount of cortisone required by the patient would be effected.

On July 13 red cell count was 3,530,000, hemoglobin 10.0 grams, hematocrit 33 per cent, platelets 92,000, and reticulocytes 15 per cent. Splenectomy was done on July 15 with removal of a 1,500-gram spleen and two small accessory spleens. At the time of operation 1,000 cc. of blood was given.

Improvement was rapid and spectacular after splenectomy. Red cell count had increased to 4,590,000 by July 20, hemoglobin was 13.8 grams (90 per cent), hematocrit 44 per cent, platelets 180,000, reticulocytes 2.9 per cent. Cortisone was discontinued. The patient was discharged on August 12 with marked clinical and hematologic improvement. He was seen again on November 3, at which time the red cell count was 3,920,000, hemoglobin 12 grams (72 per cent), platelets 190,000, white blood count 14,200 with 81 per cent lymphocytes, and reticulocytes 0.7 per cent. A hematinic was prescribed. The patient's count continued to drop, and on December 29 it was 2,030,000, hemoglobin 8 grams (48 per cent), reticulocytes 5.6 per cent, and fecal urobilinogen 115 mg. At that time 1,000 cc. of blood was given.

On January 28, 1954, his hemoglobin was 6.4 grams (38 per cent), serum bilirubin indirect 4.5 mg. Cortisone, 75 mg. per day, was started, and some improvement in the blood picture was noted by February 19. However, his hemolytic anemia seemed to be recurring. Cortisone was increased to 100 mg. per day, and 500 cc. of blood was given. On March 22 hemoglobin was 14 grams and Coombs' test was positive for the first time.

He felt well until April 4, when he was admitted with pain in the right chest, temperature of 103 degrees, and x-ray signs of pneumonia involving the

right upper lobe. He was treated with antibiotics. Surprisingly enough, his hemoglobin, which was 11.1 grams on admission, increased to 13.6 grams by April 20, with a red count of 4,500,000. At that time his lungs were clear, he was afebrile, and he was feeling fine.

This patient presents a difficult problem in management. His hemolytic anemia appeared 5 years after the diagnosis of chronic lymphatic leukemia. He rapidly reached the point where the number of transfusions required to maintain his red cell count was prohibitive, and only continued administration of extremely large doses of cortisone was at all effective. Splenectomy gave him at least a nine months' remission, and the possibility of continued control appears encouraging.

SUMMARY

Patients with leukemia and leukosarcoma may at any time during the disease process develop a syndrome of hemolytic anemia or cytopenia related to the splenomegaly accompanying these clinical states as a secondary or symptomatic hypersplenism.

Incomplete auto antibodies of many types have been demonstrated in patients with acquired hemolytic anemia. Actual destruction of antibody sensitized red cells has been found to be by intravascular hemolysis, intravascular agglutination, with mechanical trauma and lytic action on the vulnerable red cell clumps, sensitization by antibody with subsequent sequestration and hemolysis or phagocytosis in spleen and other organs.

Clinical findings in secondary hemolytic anemia, in addition to those of the primary disease, are indications of increased red cell production and simultaneous excessive destruction. There is a rapid breakdown of transfused red cells. The direct or indirect Coombs' test, and reaction with trypsin treated red cells or normal Group O red cells under certain conditions may demonstrate iso-antibodies.

Transfusions are useful in most cases, but transfusion alone is of limited value. ACTH and cortisone have been used with favorable results, but response is uncertain and unsustained in most cases, and continued steroid therapy is usually necessary to maintain remission. Since 1940, good results have been reported in the majority of cases following splenectomy, when hypersplenism is the criterion for operation. Bone marrow hyperplasia is a prerequisite, and a trial with ACTH or cortisone before splenectomy is indicated.

Two cases are presented to illustrate problems encountered in this hematologic complication of leukemia and leukosarcoma.

BIBLIOGRAPHY

1. Doan, C. A.: Hypersplenism, Bull. N. Y. Acad. Med. 25:625, 1949.

2. Doan C. A., and Wright, C. S.: Primary congential and secondary acquired splenic panhematopenia, Blood 1:10-26, 1946.

3. Wiseman, B. K.: Doan, C. A., and Wilson, S. J.: Present status of thrombocytopenic purpura with special reference to diagnosis and treatment, J.A.M.A. 115:8-13, 1940.

4. Dameshek, W.: The spleen and hypersplenism, N. Y., Grune and Stratton, Inc. 1947.

5. Boorman, K. E.; Dodd, B. E., and Loutit, J. F.: Hemolytic icterus, congenital and acquired, Lancet 1:812, 1946.

6. Coombs, R. A.; Mourrant, A. E., and Roce, R. R.: Detection of weak and incomplete Rh agglutinins, A new test, Lancet 2:5. 1945.

7. Wright, C. S.; Dodd, M. C.; Bouronde, B. A.; Doan, C. A., and Zollinger, R. M.: Studies of hemagglutinis in hereditary spherocytoses and acquired hemolytic anemia, J. Lab. Clin. Path., 1951.

8. Rosenthal, M. C.; Dameshek, W., and Burkhardt, R.: Trypsin modified erythrocytes, Am. J. Clin. Path. 21:635, 1951.

9. Dodd, M. C.; Wright, C. S.; Baxter, J. A., and Bourande, B. A.: The immunologic specificity of antiserum for trypsin treated RBC's, and its reactions with normal and hemolytic anemia cells, Blood 8:6, 1953.

10. Dameshek, W., and Schwartz, S.: Hemolysins as the cause of clinical and experimental hemolytic anemias with particular reference to the nature of spherocytosis and increased fragility, Am. J. M. Sc. 696:769. 1938.

11. Zinkham, W. H., and Diamond, L. K.: In-vitro erythrophagocytosis in acquired hemolytic anemia, Blood 7:592, 1952.

12. Muratore, L., and Cervellera, G.: The role of incomplete autoantibodies in the production of experimental hemolytic anemia induced by small doses of hemolytic serum, Acta. hematology 10:239, 1935.

13. Dameshek, W.: Acquired hemolytic anemia, Proc. of Int. Soc. of hematology, p. 120, 1950.

14. Day, R., and Perry, E.: Intra vascular hemagglutination, Blood 5:1114, 1950.

15. Castle, W. B.; Ham, T. H., and Shen, S. C.: Observations on the mechanism of hemolytic transfusions reactions occurring without demonstrable hemolysin, Am. J. Physiol. 63:161, 1950.

16. Brandt, N. E.; Bass, J. A.; Dodd, M. C., and Wright, C. S.: Phagocytosis of normal sensitized and trypsinized RBC's by tissue culture macrophages, Fed. Proc. 11, No. 1, 1952.

17. Wasastjerma, C.: Immune hemolytic mechanisms in vivo, Blood 8:11, 1042 (Nov.) 1953.

18. Dacie, J. V., and DeGruchy, G. C.: Auto antibodies in acquired hemolytic anemia, J. Clin. Path. 4:253, 1951.

19. Wagley, P. F.; Shen, S. C.; Gordner, F. H., and Castles, W. B.: The spleen as a source of a substance causing agglutination of the RBCs of certain patients with acquired hemolytic jaundice by an antihuman serum, rabbit serum Coombs), J. Lab. and Clin. Med. 35:119, 1948.

20. Loutit, J. F., and Mollison, P. L.: Haemolytic icterus, J. Path. and Bact. 58:711, 1946.

21. Dacie, J. V.: Acquired hemolytic anemia with special reference to the antiglobulin fraction, Blood 8:93 (Sept.) 1953.

22. Wernstein, J. M., and Le Roy, G. U.: Radioactive sodium chromate for the study of survival of RBCs, J. Lab. and Clin. Med. 42:368, 1953.

23. Craig, A. B.; Waterhouse, C., and Young, L. E.: Autoimmune hemolytic disease and cryroglobulinemia associated with chronic lympholeukemia, Am. J. Med. 13:6, 793 (Dec.) 1952.

24. Rosenthal, M. C.; Komninos, Z. D., and Dameshek, W.: Multiple antibody formation in autoimmune hemolytic anemia, N. Eng. J. Med. 248:537 (Mar.) 1953.

25. Young, L. E., and Miller, G.: Differentiation between congential and acquired forms of hemolytic anemia, Am. J. of Med. Sci. 226:6 (Dec.) 1953.

26. Bonney, C. W.: Primary malignant tumors of spleen with report of case of lymphosarcoma, J. Lab. and Clin. Med. 26:630, 1941.

27. Metzner, W. R., and Jones. T. E.: Primary splenic sarcomas of Hodgkins type, Cleveland Clin. Quart. 15:18, 1948.

28. Campbell, H. E., and Lubchenco, A. E.: Primary splenic sarcoma with case report, Surgery 26:847, 1949.

29. Hogan, P. S., and Watson, C. J.: Hypersplenism and hemolytic anemia in leukemia, results of splenectomy, Proc. 3rd Internat. Soc. Hematol. N. Y., Grune and Stratton, 1951.

30. Evans, T. S., and Doan, C. A.: Hemolytic crises of acquired hypersplenism in coarse of giant follicle lymphoma, Proc. 3rd. Internat. Cong. Int. Soc. Hemat. Grune and Stratton, New York. p. 102, 1951.

31. Aas, K.: Splenic hemolytic anemia. Treatment by splenectomy, Acta Chirurg. Scand. 104:81, 1952.

32. Sacks, M. S.; John, E. F., and Workman, J. B.: Diagnosis and treatment of acquired hemolytic anemia, J.A.M.A. 150:16 (Dec. 20) 1952.

33. Fisher, J. H.; Welch, E. S., and Dameshek, W.: Splenectomy in leukemia and leukosarcoma, New Eng. J. Med. 246:477, 1952.

34. Dameshek, W.; Rosenthal, M. C., and Schwartz, L. J.: The

treatment of acquired hemolytic anemia with ACTH, New Eng. J. Med. 244:117, 1951.
35. Gardner, F. H.; McElfresh, A. E.; Harris, L. W., and Doan, W.: The effect of ACTH in idiopathic acquired hemolytic anemia as related to the hemolytic mechanisms, J. Lab. and Clin. Med. 37:444, 1951.
36. Feldman, J. O.; Rachmilewitz, M.; Stein, O., and Stein, Y.: The adrenal cortex and hemolysis, Blood 8: 4,342 (April) 1953.
37. Saint, E. G., and Gardner, H. J.: The successful treatment of severe acquired hemolytic anemia with adrenocortotropic hormone, Med. J. Austral. 2:305 (August 30) 1952.
38. Garcia, J. F.; Van Dyke, D. C.; Huff, R. L., and Elmlinger, P. J.: Increase in circulating red blood cell volume and hypophysectomized rats after treatment with ACTH, Proc. Soc. Exper. Biol. and Med. 76:707, 1951.
39. Frelick, R. W., and Sluzar, O.: Treatment complications in a case of lymphatic leukemia with secondary hemolytic anemia, Delaware Med. J. 25:83 (April) 1953.
40. Zollinger, R. M.: The surgical management of hypersplenism, Missouri Med. 50:193 (March) 1953.
41. Meyers, M. C.; Miller, J. W.; Linman, J. W., and Bethel, F. H.: Use of ACTH and cortisone in idiopathic thrombocytopenic purpura and idiopathic acquired hemolytic anemia, Annals of Internal Med. 37:253 (August) 1952.
42. Witts, L. J., et al.: Report to the medical research committee on treatment of blood disorders with ACTH and cortisone, Brit. Med. J. 2 (December 26) 1953.
43. Aitchison, J. D.: Hemolytic anemia treated with ACTH, cortisone and splenectomy, Brit. Med. J. 1:4801, 1953.
44. Etess, A. D.; Bassen, F.; Litivins, J., and Sussman, L. N.: Acquired hemolytic anemia treated with ACTH prior to splenectomy, Acta Haem. 6:8-2, 1951.
45. Cochrane, H. A., and Gross, L.: Late lymphatic leukemia complicating hypersplenic syndrome, Arch. Int. Med. 9:1-82 (Jan.) 1952.
46. Morrison, M.: Samwick, A. A.; Ruhinstein, J.; Swiller, A. J., and Loewe, L.: Spleen aspiration in osteosclerosis and splenic hematopoesis (Rationale of splenectomy), Acta Hemat. (Basel) 6:309, 1951.
47. Ferrata, A., and Fieschi, A.: Spunti di clinica e terapia delle splenopatie haematologica, 20:1, 1939.

Arrhenoblastoma

G. E. Kassebaum, M.D.

El Dorado, Kansas

Arrhenoblastoma of the ovary presents one of the dramas of medicine, and its apparent rarity leads us to report two cases which occurred in our relatively small hospital within a period of 10 years. G. Barzilai[1] reported 50 cases on record up to 1949. C. Y. Thomas et al.[2] reported 121 cases up to 1951.

There has been much confusion in the nomenclature of this tumor, i.e. adenoma tubulare carcinomatosum, fibroblastic sarcoma of ovotestis, carcinoma, and endothelioma. The most generally accepted name now is arrhenoblastoma, so designated by R. Meyer, who first identified the varied patterns of this tumor as belonging to a special oncologic group.

Histogenesis of arrhenoblastoma is still unsettled. R. Meyer's theory, probably the most accepted, suggests that the early development of the ovary is identical with the testis. In the later stages of female differentiation, certain elements of male differentiating potency may be left in the medullary portion of the ovary. Another theory is that arrhenoblastoma always arises in an ovotestis and is based on actual observation of one case (Barzilai). Others feel that the tumor arises from remnants of the rete testis.

Arrhenoblastomata occur in very young as well as in old patients, perhaps most commonly in the 20- to 30-year group. Our younger was 14 years of age and the older 56. Meyer reported one at 60 years.

The tumor is usually unilateral. It may retain the shape of the ovary or be rounded, lobular, or smooth. The outer surface is bluish or yellowish in color. The size varies from that of a pea to a large grapefruit, and the tumor may entirely replace the ovarian tissue. Mostly solid, the tumor may contain small cysts filled with serous or bloody fluid. Small tumors are usually hard and the larger ones soft, due to necrosis and hemorrhage. On cut surface the tumor shows mottled areas of bluish-purplish-yellowish hues. The tumor does not adhere readily to adjacent structures. Coincident atrophy of the uterus and opposite ovary is usually present. Extension to other parts of genital organs or peritoneum is rare, except in some malignant cases.

The microscopic picture is usually polymorphocellular. The following cellular structure may appear in varied combinations:

1. Spindle shaped or rounded cells resembling early undifferentiated cell type of the early mesenchymal core of the gonads.
2. Large spherical cells resembling granulosa cells.
3. Cuboid or low columnar cells resembling Sertoli's cells of the male gonad. Neither sperm cells nor any of their progenitors have been found.

Arrhenoblastoma is a hormonally active tumor. This is proved by the marked signs of virilization which regress after its removal and reappear if the tumor recurs. Symptoms due to its hormonal activity give the tumor its distinctive position among ovarian neoplasms.

The first changes to appear are always those of de-feminization: cessation of menses, sterility, and flattening and atrophy of breasts. Later follow the masculinizing signs. The figure loses its normal female curves and assumes the more angular male conformation. Masculine hair distribution develops over the body and on the face. In progressive cases, a true beard and baldness have been known to occur.

* El Dorado Clinic, El Dorado, Kansas.

Acne is frequently present. The expression of the face becomes masculine. The voice becomes deep and coarse. The ligaments and cartilages of the larynx appear enlarged. The clitoris hypertrophies but the sexual impulse remains feminine.

Almost all signs and symptoms disappear after the removal of the tumor and usually do so in the order of appearance. Changes in the larynx and clitoris are usually irreversible.

Arrhenoblastoma is classified as a malignant tumor but is far less so than other epithelial tumors. Simple surgical removal usually effects a cure. If, however, symptoms do not regress following surgical extirpation of the tumor, metastasis has probably occurred and the outlook is grave. Irradiation should be carried out in all cases where pathologic study suggests marked malignancy.

CASE REPORTS

Mrs. K. H., 56, nullipara, was admitted July 1, 1942, complaining of a bearing down sensation in the pelvis and perineum. Her menstrual life had been regular and had ceased eight years before. She reported, however, that four months earlier she had menstruated for several days, an occurrence which was repeated twice later. Hirsutism of face and abdomen was markedly increased, and some huskiness of the voice was noted.

The pathological report following dilatation and curettage indicated low grade carcinoma of the fundus. She was given x-ray therapy on July 2, 1942, followed by a total hysterectomy, including tubes and ovaries. The left ovary, the size of an apple, was found to be hard and nodular; the right ovary was atrophied; the fundus was small with multiple calcified nodules; the cervix was normal. The pathological report was arrhenoblastoma, left ovary.

Miss B. B., 14, nullipara, was admitted February 13, 1952. Although she may have been a little early in maturing, she showed normal physical development. Menses, beginning at 11 years, had been normal until six months previously, when amenorrhea began, with light spotting at times. Three months before admission the patient suffered an acute sore throat, and hoarseness had been persistent since that time.

Physical examination disclosed an abdominal tumor which was gradually enlarging and an increased growth of hair on her upper lip. An ear, nose, and throat examination showed bridging of cords as found in chronic laryngitis.

A laparotomy was performed February 14, 1952. A multilocular, semi-solid and cystic tumor about the size of a grapefruit was filling the lower abdominal cavity. The pedicle of the tumor was crushed and cut. The remainder of the right tubo-ovarian ligament and the right fallopian tube were excised. The left tube and ovary were normal and were left intact.

Following is the pathological report:

Macroscopic examination: The specimen consists of a 6- or 7-inch cystic mass of ovarian tissue presenting many multiloculate cavities, some areas of gelatinoid yellowish tissue. There is also a 5 x 60 mm. appendix.

Microscopic examination: Diffuse fibrosis is revealed throughout the ovary with irregular cord-like masses of cells which have a pinkish cytoplasm of moderate amount. The nuclei vary in size and in staining characteristics, generally are ovoid, moderately dark staining. At times the cytoplasm is box-like in character. In areas, these cells are forming an irregular lacy mesh work with a number of glandlike spaces lined by cubical epithelial cells of moderate height. There are some areas of fibrosis, and, in some instances, the cells grow in cord-like masses. They appear to be malignant but well within the confines of the tissue. In view of the type of growth, the grade of malignancy remains questionable. These tumors appear malignant but behave in a benign manner. This, however, may be considered as a malignant growth, at least from the microscopic appearance. The capsule of the ovary appears to be relatively intact, but the growth characteristics are otherwise those of a malignant process. The diagnosis is arrhenoblastoma, a malignant neoplasm.

Four months later the menses returned. Gradually the hirsutism disappeared, but two years after operation the voice remains hoarse, which is usually true in these situations. The patient remains in good health and apparently there are no metastases.

BIBLIOGRAPHY

1. Barzilai, G.: Atlas of ovarian tumors, Grune and Stratton, New York, 1949.
2. Thomas, C. Y.; Fisher, E. R.; Turnbull, R. B., and Krieger, J. S.
3. Arrhenoblastoma of the ovary, Ann. Surg. 135:4, 543 (Apr.) 1952.
4. Cancer, Ackerman and Regato, C. V. Mosby Company, St. Louis, 1947.
5. Douglas, Gilbert F.; Douglas, George Capers, and Douglas, Gilbert F., Jr.: Tumors of the ovary, 41:5, 567 (Nov.) 1951.

I know of no safe depository of the ultimate powers of society but the people themselves, and if we think them not enlightened enough to exercise their control with a wholesome discretion, the remedy is not to take it from them, but to inform their discretion by education.—*Thomas Jefferson.*

THE MONTH IN WASHINGTON

Editor's Note. The following summary of Washington news was prepared by the Washington office of the A.M.A. for distribution to state and regional medical journals.

With the change in control of Congress, there naturally will be a major reshuffling of all committees, including those handling medical and health legislation. A new chairman moves to the top, and at the bottom a few Republican members drop off, to be replaced by an equal number of Democrats. In a Congress so evenly divided, domination of this committee machinery is a vital asset.

A majority of the Democrats taking over committee chairmanships in January will be returning to the same jobs they held when their party was in power, but the situation is a little different on the two committees most important in health and medical legislation. It will be the first time either of these chairmen has had the responsibility of running the full committee, although both have been involved in medical legislation for many years. Both are veteran legislators and are Southerners. They are Senator Lister Hill of Alabama, who replaces Senator H. Alexander Smith of New Jersey as chairman of the Labor and Welfare Committee, and Rep. Percy Priest of Tennessee, who succeeds Chairman Charles Wolverton, also of New Jersey, on the Interstate and Foreign Commerce Committee.

By reason of seniority, Senator James Murray of Montana is in line for the Labor and Welfare Committee chairmanship. However, he has announced that he prefers to run the Interior and Insular Affairs Committee, thus turning over the other chairmanship to Senator Hill. Senator Murray, as a sponsor of national compulsory health insurance, and as a chairman and member of its committee that held such turbulent hearings on this subject, became well known to the medical profession.

Senator Hill, the son of a physician, has been in Congress for 30 years—14 in the House before he came to the Senate. He was a co-sponsor of the Hill-Burton hospital construction program, perhaps the most important piece of medical legislation enacted since World War II.

Presumably the Senate committee's Health Subcommittee again will be headed by Senator Herbert Lehman of New York, who handled this task during the last Democratic Congress, the 82nd.

Mr. Priest is a former school teacher and newspaperman. He has been in the House for seven uninterrupted terms. In 1951 he was chairman of the Commerce Committee's Health Subcommittee; the subcommittee system was abolished by the committee in 1952. Since then he has taken an extremely active part in committee work in health and medical fields.

The Hill and Priest committees will handle most health legislation with the exception of military, veteran, and appropriation bills. For example, they will be in charge of reinsurance if it is reintroduced, as well as most health-medical bills originating in the Department of Health, Education and Welfare.

A number of other committee changes of importance to medical legislation are scheduled. Rep. Edith Nourse Rogers of Massachusetts, a veteran of 29 years in the House, loses the chairmanship of the Veterans Affairs Committee. She is being succeeded by Rep. Olin Teague of Texas, who was elected to Congress for the first time while he was completing his six-year Army duty in 1946.

The House Appropriations Committee chairmanship goes from Rep. John Taber of New York to Rep. Clarence Cannon of Missouri; both have the reputation of being economy-minded. Of considerable significance in medical appropriations is the change in the chairmanship of the subcommittee that handles money for the Department of Health, Education and Welfare. The chairman for the last two years, Rep. Fred Busbey of Illinois, carefully scrutinized all health appropriations and effected many reductions. He was defeated for re-election. The prospective chairman of the subcommittee, Rep. John Fogarty of Rhode Island, repeatedly has intervened in the committee and on the House floor to restore money cut out by the subcommittee.

Chairman of the Armed Forces Committee in the Senate—where medical care for military dependents would be taken up—will be Senator Richard B. Russell of Georgia, replacing Senator Leverett Saltonstall of Massachusetts. On the House side, the Armed Forces chairmanship goes to the veteran Rep. Carl Vinson, also of Georgia. He replaces Rep. Dewey Short of Missouri.

Any bills proposing reorganization of the executive departments will come before Chairman John L. McClellan of Arkansas in the Senate and Rep. William L. Dawson of Illinois in the House. They are succeeding Senator Joseph R. McCarthy of Wisconsin of Rep. Clare B. Hoffman of Michigan.

(This summary is, of course, predicated on the Democrats surviving a threatened recount of the close Senate vote in Oregon and going ahead with organization of both chambers.)

Plan now to attend the 96th annual session of The Kansas Medical Society, Hutchinson, May 1-5, 1955.

PRESIDENT'S PAGE

Within a few days we will have entered 1955 when problems relating to the physical well being and the health of people will again be spread before our legislative chambers.

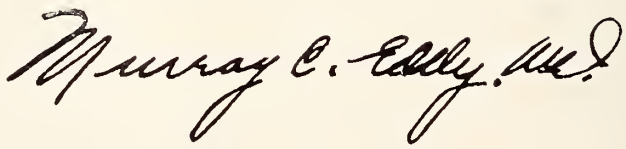
At the federal level, Congress will deliberate a reinsurance program and a hundred other items that will in one way or another influence the health of the American people. Kansas will once more determine the minimum education requirements for physicians.

We have collectively elected persons to speak for us on these and other issues. We will now be governed by their wisdom. If we hold a conviction upon an issue and have experience to support our belief, we are morally obligated to express our view. As citizens, we owe elected officials the benefit of our special knowledge in the field of health.

In this coming fateful and decisive year, it is my earnest hope that you will each fulfill your very special role in giving the public the highest possible quality of health care. This implies not only your professional services as a physician but your duty as a citizen.

Therefore, the New Year's resolution for myself will be just that, and with the heartiest and most cordial greetings on this occasion for you and for your family, permit me to add the sobering reflection that we must each participate in this coming venture if we shall continue to give the public the benefit of sound and progressive health legislation.

Yours,



EDITORIAL COMMENT

New Year's Resolutions

Next year will be different. My New Year's resolutions are already made to give me a running start before the scoring begins. There will be just five which for some unaccountable reason all begin with the letter c, perhaps because they go into effect on Christmas eve. Here they are.

I will be curious. During the year I shall prepare two scientific papers, one for oral presentation and the other for publication. This is not vanity on my part but a deliberate effort to organize my thinking upon two separate subjects, to perform a mental exercise for the benefit I personally will receive thereby.

I will be critical. For the coming year I intend to be increasingly critical of organized medicine at all its levels. I plan to discover what medical organization means and how it functions. I shall not one year longer be led unknowing and unwilling into paths I cannot see. So, speaking for one member, I intend to learn what my society hopes to accomplish and shall then exert an honest effort to either change its course or serve its purpose. So I will be critical and zealous in my effort because I alone am personally involved in what succeeds or fails.

I will be considerate. Here my confidence begins to waver because I find myself at the threshold of an indefinable area. Perhaps the subject is ethics. It certainly relates to professional conduct. Of course, we all intend to render the best possible professional care, but I have something else in view. I want to be considerate of my patient, not only as a patient but as an individual who in his mind is the most important living person. If that means trouble, at least it is my own. And I have elected to assume its consequences.

I will be a citizen. I will analyze my obligations to my country, state, and to the town in which I live. Once this reflection has been completed, I propose to abandon my present lethargy and undertake not less than one project, unrelated to my profession, which I may actively pursue with honesty and with enthusiasm to completion.

There is one more, another c. I know, it was the earnest resolution for this one year at least to wish you each a Merry Christmas before the holiday has passed. So I shall do so now and, whatever happens to the other four, one resolution will have been carried to reality, and even on that basis alone the next year will be different.

Federal Aid in Kansas

Part III

This portion of the story of federal aid in Kansas will briefly discuss several areas, again without critical comment. The series is intended to provide the physician with factual information rather than to discuss purposes or values.

EDUCATION

Federal aid for educational purposes is among the larger sums obtained in Kansas. There are five programs in this field, three of which are administered by the superintendent of public instruction and the last two are operated by the Vocational Education Department.

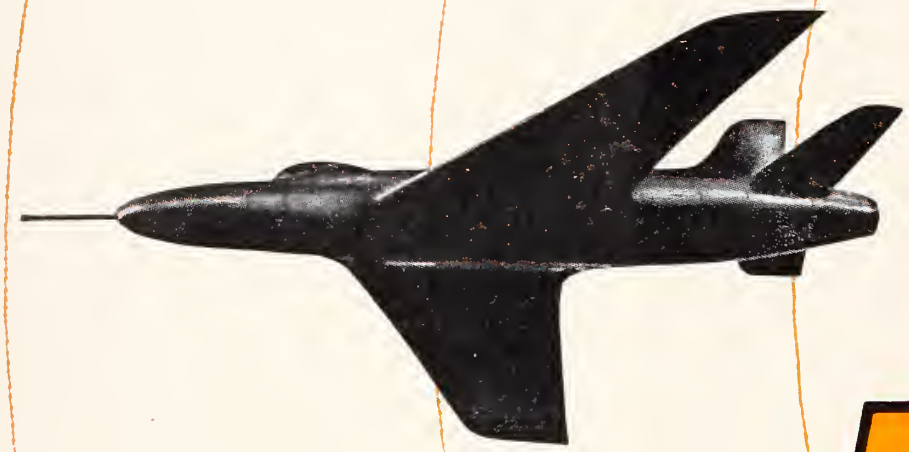
The national school lunch program was authorized by Congress in 1946. It is federally administered by the U. S. Department of Agriculture. The Superintendent of Public Instruction directs the program for the state and the Board of Education locally. The formula is involved, taking into account the school census, the per capita income as compared with the national figure, etc. Federal aid has been relatively constant over the years and in 1953 was $788,900. The state paid $26,585 and local sources contributed $3,341,132 for a total of $4,156,617. In Kansas, 1,086 schools participated in this program.

The next two items are emergency grants, first for school construction and second for maintenance and operation of schools in areas upon which the federal government has placed financial burdens by reason of civilian or armed forces activities. This is a little involved because of the variety of coverage, so an analysis will not be attempted here except to say that the federal government gave Kansas $2,600,000 for this purpose in 1953 and Kansas paid $10,000.

VOCATIONAL WORK

There are two programs for which Vocational Education last year received $366,810.26 from federal sources, $250,000.37 from the state, and $789,108.94 locally for a combined cost of $1,405,919.57. This is about constant over the years except that the federal grant is decreasing slightly and local participation expanding. The money is used for instruction, teacher training, etc. A sizable portion of the cost goes for veterans' training. There have been 11,156 veterans enrolled in this program.

Vocational rehabilitation in Kansas has received federal aid since 1937. This is all operated by federal and state funds with no local participation. The formula is simple. The federal government pays the entire cost of administration, the entire cost of vocational guidance and placement services for each state, and half the cost, as long as there are funds, for rehabilitation training, tools and equipment, books,



accepte

ACH

record time

ROMYCIN*

Hydrochloride
Tetracycline HCl Lederle

ACHROMYCIN, new broad-spectrum antibiotic, has set an unusual record for rapid acceptance by physicians throughout the country. Within a few months of its introduction, ACHROMYCIN is being widely used in private practice, hospitals and clinics. A number of successful clinical tests have now been completed and are being reported.

ACHROMYCIN has true broad-spectrum activity, effective against Gram-positive and Gram-negative organisms, as well as virus-like and mixed infections.

ACHROMYCIN has notable stability, provides prompt diffusion in body tissues and fluids.

ACHROMYCIN has the advantage of minimal side reactions.

LEDERLE LABORATORIES DIVISION *AMERICAN Cyanamid COMPANY* Pearl River, New York

Lederle

*REG. U.S. PAT. OFF.

living expenses during rehabilitation, medical treatment, hospitalization, and prosthetic devices. The program is open to physically or mentally handicapped persons over 16 years of age and is designed to render them fit for remunerative employment. Last year Kansas spent $276,718.59 for this program, of which $184,866.50 was federal money and $91,852.09 state.

HIGHWAYS

The Federal Aid Road Act was passed by Congress in 1916. Kansas has participated in this program since 1917. It is federally administered by the U. S. Department of Commerce, at the state level by the Highway Department, and locally by the county commissioners. Grants are based on population, land area, and post road mileage of the state compared with United States totals and are generally on a 50-50 basis. Figures are available for all types of roads, but the total only is given here. In 1953 the federal government gave Kansas $12,573,566 for roads. The state spent $10,247,558, and local sources spent $3,119,686 for a total of $25,940,810. So federal aid in this category is about half the expenditure and, considering federal grants to all states totalling $575,000,000, Kansas received 2.1 per cent.

LABOR

The only item listed pertains to unemployment compensation and employment service. Federal participation is limited to administrative costs of the state agencies. The fund is originally raised by a three per cent tax on all payrolls with eight or more employees. Actually this program has changed recently, but the 1952 report is the latest available. Three tenths of one per cent of this tax is forwarded to the general treasury of the United States, so Kansas keeps 2.7 per cent for local allotments. The federal government pays all administrative costs of the offices conducting these services which, for Kansas at least, have never equalled the amount sent in. In 1952 Kansas paid the federal government $2,325,000, representing three tenths of one per cent of the three per cent tax. The government financed a state and 26 local employment offices at a cost of $1,435,200, which represents a net loss for that year of $889,800.

The fourth in this series, to appear next month, will deal with the welfare program.

A Message From the Auxiliary

Yuletide greetings to you from the Woman's Auxiliary to The Kansas Medical Society!

The co-operation, understanding, and helpfulness of your president, Dr. Murray C. Eddy, have been a wonderful Christmas gift to us lasting the year through. We are also most grateful to the Advisory Committee of The Kansas Medical Society for the approval of our goals and programs and for the many inspiring and constructive suggestions to us.

I wish to take this opportunity to express some thoughts on our mutual problems.

The magazine *Today's Health* concerns all of us. The complimentary six months' subscription sent to your office will soon expire. Won't you send in your subscription fee of $1.50 to Mrs. Chester Young, 4315 Victory Drive, Kansas City, Kansas. Through "Operation M.D." it is possible to have *Today's Health* in the waiting room of every doctor in the United States. If this magazine could be self-supporting by an increase in circulation and advertising, the deficit made up each year by the A.M.A. would be available for other needed programs.

The membership of the Auxiliary has been foremost in my thoughts. The Kansas Medical Society and our group form a partnership. Every doctor should be proud to have his wife as a member. Enroll her so she may assist the Auxiliary in its program for the advancement of medicine, public health, and public relations. State and national dues are $2.00. Send your check to Mrs. Louis Graves, St. John, Kansas.

Civil defense is one of our problems. All of us agree that America will need more effective civil defense if we are going to brace this country for the shock of a possible attack with modern weapons and survive that attack with enough people and productive facilities to go on and win a war. Doctors have adopted the philosophy of mutual aid and reciprocal assistance. Encourage your wife to work with her Auxiliary so she may aid the medical profession in meeting responsibilities and in spreading its philosophy to everyone. The reading of Philip Wiley's book *Tomorrow* should jar any person from the spirit of complacency.

Our Auxiliary believes that a deterioration of our medical training standards would be a national disaster. We are giving our wholehearted support to the American Medical Education Foundation. Each of our 2,000 members has been asked to contribute $1.00 to this foundation.

Doctors realize more than we the need for more nurses. To help recruit girls for this vocation, we have established an extensive loan program. Additional encouragement is given by local auxiliaries and hospitals. Twenty-six counties are actively engaged in nurse recruitment programs. Fifteen Future Nurse Clubs have been formed with auxiliaries serving as sponsors. Their membership is approximately 500 girls.

May we remind you of our Auxiliary Memorial Fund for nurse scholarships. If anyone wishes to give a memorial in the name of a deceased Auxiliary member or friend, money may be sent to Mrs. Rich-

ard O'Donnell, Ellsworth, Kansas. She in turn will send a card to the family of the deceased.

Lately I have deliberated on the resolution "A Doctor for Every Doctor's Family." The need of a personal physician for the doctor and his family should be heeded. The medical profession has a higher death rate than that of any other profession in our country. It is exigent that doctors regard with care the advice given them by their family physician. Doctors must live longer; they are needed. A strong healthy doctor is a wonderful advertisement to the medical profession.

All that our Auxiliary does is done to aid the medical profession. At any time, call on us to help you. Never underestimate the power of your Auxiliary.

At this holiday season may the spirit of Christmas be in your heart and bring you joy all the year through.

—Elizabeth Millis, President
Woman's Auxiliary to The Kansas Medical Society

P.S. As a reminder—more people die in traffic on December 24 than on any other day of the year. "Don't give your life for Christmas."

Age and Distribution of Doctors

A recent study of the age and distribution of Kansas physicians has revealed a number of interesting facts. This survey includes reports on 1,816 physicians in Kansas.

Effects of the Kansas Plan, originated in 1948, may be realized by a study of the over-all picture. In 1948 the average age of the doctor in Kansas was 54.6. Today, or after five years, the average is 49.8 as a result of the influx of younger doctors in Kansas. The median age likewise dropped proportionately, and today that age is 46. The oldest member of the Society is 98 years of age and the youngest is 25. There are 364 members of the Society under 36 years of age.

Information on doctor distribution in 1948 is not available. The records today show 79 physicians practicing in 68 Kansas towns with populations under 500. One hundred twenty-two are located in 87 communities with populations ranging between 500 and 1,000, making a total of 201 practicing in towns having fewer than 1,000 residents. Cities having populations of from 1,000 to 5,000 persons receive services from 480 physicians. Ten communities in Kansas have populations numbering between 5,000 and 10,000, and 146 physicians have chosen to locate in those cities. The remaining physicians in the survey, 989, practice in cities of more than 10,000 residents.

The Executive Office of The Kansas Medical Society maintains a locations list for the aid of communities in need of physicians and physicians seeking locations.

BLUE SHIELD

The presence of participating physicians at most of the district Member Council meetings this fall was enthusiastically received by the members.

All members of Kansas Blue Cross-Blue Shield are privileged to participate in activities of the plans by expressing their opinions and wishes through the organization known as Blue Cross-Blue Shield Member Council. District council meetings are held twice each year.

Member Council is a representative group of members who speak independently of the plan. It is organized on the county, district, and state level; it is the "voice of the members," which trustees and staff listen to and consider seriously in planning the best possible benefits for Kansas people.

One-third of the Blue Cross Board of Trustees is composed of lay members. Each of the nine Member Council districts elects a member to the board. The remainder are elected by the state Members Committee. The chairman of the state Members Committee is also one of three lay members on the Blue Shield board.

The active participation by Blue Shield doctors in these meetings has done much to stimulate interest and create understanding between the member and the medical profession. The objective discussion of community health problems takes on new meaning to the member when a doctor lends professional opinion to the meeting. At the same time, public opinion is accumulated by the profession.

Success of these meetings suggests that, at some future date, it might be desirable to hold district meetings involving all groups of the Blue Cross-Blue Shield program, the doctors, the hospitals, and the members. Through this type meeting a mutual understanding of health needs and solutions could, perhaps, best be attained.

Medical Education Expands

Medical education has expanded considerably since 1945, according to a recent study made by representatives of the Association of American Medical Colleges. During the period covered, three medical schools have opened, the University of Washington, the University of California at Los Angeles, and Miami. Two, Alabama and North Carolina, have expanded to a four-year curriculum.

Biopsy Procedures in Office Practice

Tumor Conference

Edited by Irwin Joffe, M.D.

Dr. Helwig: Biopsy as a diagnostic technic was introduced by the German pathologist, Ruge,[1] in the 1800's. Between that time and the start of the present century, it fell into disrepute largely due to the inadequate training of pathologists, who often misinterpreted what they found in the tissues examined. For the last 50 years, however, biopsy has been a standard procedure. We should recall that no cancer, except under rare circumstances, should be treated before a positive diagnosis by biopsy has been established. The literature and the experience of all of us are replete with instances in which the patient has been mistreated because a biopsy was lacking or was wrongly interpreted.

I can't emphasize too strongly that when you send a specimen to your favorite pathologist you accompany it with every bit of pertinent data available. This would include the sex, age, and race of the individual, the duration, location, and gross character of the lesion, and history of previous biopsy or treatment. The pathologist's morphologic interpretation must frequently be guided and modified by clinical information, which allows him to study tissues in the perspective of their relationship to the general status of the patient. You are doing your patient no service, and you are doing yourself no service, and you are doing the pathologist distinct disservice if you don't give him every bit of data which you have at hand.

I'm going to ask Dr. Robinson to tell us about some of the pitfalls to avoid in the taking of biopsies.

Dr. Robinson: In general, when dealing with a large, accessible neoplasm, one should excise with the cold knife a small piece of the viable margin of the tumor along with a little of the adjacent normal tissue. A small tumor may often be managed best by excision biopsy of the entire lesion. Perhaps some of the greatest difficulties are encountered in the deep biopsy. The physician who attempts such a biopsy through a small incision is likely to come up with something less than the tumor. Such a biopsy gives no information and may give a false sense of security.

Another common pitfall in taking a biopsy is to take the most obvious part of the tumor, the most exposed part. The sample selected may be completely necrotic, or may be composed entirely of exudate. The pathologist can't reach a diagnosis with material of this character.

Proper biopsy of lymph nodes requires care and judgment. Nodes in the region of ulcerated or recently treated lesions may enlarge as the result of inflammatory changes alone. Usually, but certainly not invariably, such inflamed nodes are less than 1 cm. in diameter. Larger lymph nodes in the drainage area of a neoplasm must be viewed with more suspicion since they will frequently contain metastatic tumor. Select the right node. Don't take a tiny node that's off to the side simply because it is easy to get; it may not give any useful information. Take a large node that's in the obvious lymphatic drainage area of the primary tumor, or one which seems obviously involved by lymphoma. Usually the entire node should be removed with capsule intact.

Beware of taking lymph node biopsies through small incisions. I'll never forget one of the first cervical nodes that I was asked to remove as a junior resident. I was working through a little nick over the front of the neck when suddenly the floodgates opened and I couldn't see or feel anything. A man of experience, the senior resident, literally bailed me out rather quickly and simply by tying off the jugular vein, which I had inadvertently opened. Always secure adequate exposure if you are going to invade an area where you can get into trouble.

Dr. Helwig: Thank you, Dr. Robinson. Dr. Proud, will you tell us something about biopsies in the hidden cavities of the head and neck.

Dr. Proud: In taking biopsies from the nose and throat, one will require an effective local anesthetic; the most effective one in my hands has been cocaine, a 5 per cent solution. One can probably avoid trouble by preparing the patient in the following manner. Perform the biopsy shortly after the patient has eaten. The incidence of hypoglycemic syncope and cocaine reaction is greater before meals. A preanesthetic dose of a barbiturate 15 minutes before the application of cocaine solution is also advisable. Use a vasoconstrictor drug locally. It will help to fix the cocaine to the tissue and give better analgesia. It will also diminish systemic absorption of cocaine. Use cocaine sparingly and accurately; wring out the applicators before applying the drug. Repeated applications of small amounts are more effective than one

Cancer teaching activities at the University of Kansas Medical Center are aided by grants from the National Cancer Institute, U. S. Public Health Service, and the Kansas Division of the American Cancer Society. Dr. Joffe is a Trainee of the National Cancer Institute.

copious application because the cilia carry much of it away.

I think the hardest area to approach for an adequate biopsy is the nasopharynx. The laryngologist often uses a palate retractor as an aid to visualizing this area. The general practitioner, however, does not need a $20 palate retractor for biopsying the nasopharynx. A couple of rubber catheters will do just as well. One of them is passed into each nostril and out of the mouth; each catheter is then held taut by clamping its two ends with a hemostat. Then, with the use of the tongue blade and the mirror, one can examine the nasopharynx satisfactorily; and, having someone hold the tongue down, one can slip a conchotome through the nose and under indirect visualization with the mirror one can see the nasopharynx and watch the biting forceps snip off a specimen of tumor. Obviously some of the rules that Dr. Robinson has laid down for us will not be strictly followed in taking biopsies from the nasopharynx. It is hard enough to get a biopsy, and it is often impossible to select accurately areas where tumor tissue is invading the normal. I'm afraid our biopsies—those of the nasopharynx particularly—are probably some of the worst that the pathologist receives.

In the nasopharynx there is one type of tumor that should never be biopsied; it is the tumor known as the nasopharyngeal fibroma.[2] This tumor is extremely vascular, and even when unmolested it has been known to bleed fatally. Every now and then some physician applies an adenotome to one with the intent of performing an adenoidectomy and becomes so fascinated in bailing out the blood that the patient exsanguinates before the physician realizes what has happened. A nasopharyngeal fibroma is smooth, an adenoid is rugose. A nasopharyngeal fibroma is purplish-red, an adenoid is pale. A nasopharyngeal fibroma is hard, whereas an adenoid is compressible. It is wise to visualize and palpate the nasopharynx before attempting a biopsy.

To take a biopsy from the nose, a nasal speculum and a conchotome are necessary; the nasal speculum will expose the nasal fossa, and the biopsy can be taken with the conchotome. Now there are two types of conchotomes or forceps. One is a cup forceps, and I know the pathologists resent it because it crushes the specimen. The other type, the punch forceps, has sharp edges and punches out a piece of tissue which is considerably less distorted.

A pack should always be available when biopsying in the nose. Brisk bleeding is occasionally provoked, and if a pack is handy it may eliminate considerable anguish. The best type of pack for controlling immediate hemorrhage is a small cotton pledget soaked in epinephrine, which is inserted promptly after the biopsy is taken. The resultant vasoconstriction will help to prevent hemorrhage. If a bulkier pack is needed over a longer period, it should be coated with petrolatum.

When physicians take tissue from other parts of the body, they send it to the pathologist to find out what it is, but it has become popular to discard structures removed from the nose or the ear; all too often they turn out to be malignant. Tissue that comes out of the ear, the nose, or the throat should be studied by the pathologist, just as other tissues.

To biopsy in the ear with any satisfaction, one has to have a very small biopsy forceps. Obviously, the size of the specimen which one will procure with such a forceps is not going to thrill the pathologist. The biopsy can be accomplished through an ordinary ear speculum or through the electric otoscope. Upon finding exuberant granular tissue in the middle ear, one is tempted simply to cauterize it to make it go away. Believe me, tumor can occur in the ear, just as it can occur anywhere else in the body; and when granular tissue is found in the ear, it definitely should be biopsied. Before manipulating a growth in the ear, one should exclude the presence of the glomus jugulare tumor,[3] a vascular neoplasm which sometimes extends into the aural cavities. This tumor is purplish-red and usually produces pulsating tinnitus which the patient likens to the sound of a locomotive; he hears a swish with each heartbeat. Such a sound can also be heard by the examiner



Figure 1. Distorted tissue of uterine cervix in biopsy secured with cautery knife (mag. x100).

upon auscultation. If gentle manipulation with a probe initiates brisk bleeding, a biopsy should not be attempted.

Biopsies of the larynx and hypopharynx are best left to the specialist. A good biopsy of the tongue must be a deep one, and a deep one quite often will lead to hemorrhage. Lesions of the gingiva in people with dentures may be due only to irritation by the dentures; if they are of recent onset, biopsy may be deferred during a trial period of 2 to 3 weeks without dentures.

Dr. Helwig: Thank you, Dr. Proud. I'd like to add one other tumor which sometimes bleeds profusely, and that's the hemangioma.

Dr. Boley, may we have your comments?

Dr. Boley: I have selected a few slides to illustrate some of the points made by Dr. Proud and Dr. Robinson. As was pointed out, the choice of instruments is most important in determining the quality of the material that reaches the pathologist and consequently influences the character of his interpretation. Select a delicate forceps to avoid crushing tissue and, above all, avoid the cutting cautery.

I would like to show a miscroscopic slide of tissue (Figure 1) that was removed from the cervix with a cautery knife. As we scan the section, we note that the heat of the cautery has distorted or destroyed every cell in the fragment. No interpretation is possible; the efforts of the referring physician and the inconvenience and expense to the patient have been in vain. If the cautery is used to achieve hemostasis, it should be applied only after the biopsy has been secured with the use of the cold knife.

If a biopsy is to be satisfactory, the quantity of tissue must be adequate. The next section I have selected to show (Figure 2) is an example of an inadequate uterine curettage. One sees desquamated cells from the vagina and small amounts of blood. The few microscopic fragments of endometrium seen are inadequate for interpretation. You must submit adequate material if you want a diagnosis.

Another item which deserves emphasis is proper fixation of biopsy material. Prompt fixation in an adequate amount of 10 per cent formalin or other fixative will prevent distortion due to drying. Lymphoid tissue is particularly vulnerable to drying and to rough handling and should be given special care.



Figure 2. Inadequate uterine curettage showing a few fragmented endometrial glands and cellular debris (mag. x100).



Figure 3a. Biopsy of vulval skin which was allowed to dry prior to fixation (mag. x100).



Figure 3b. Properly handled biopsy from adjacent site showing carcinoma *in situ* (mag. x100).

Better preservation of lymph nodes will be achieved if they are halved before being placed in fixative. In fact, half of the node may be reserved unfixed for bacteriologic cultures, a technic too often neglected. Not uncommonly, microscopic study of a lymph node discloses only a non-specific inflammatory process. Definitive diagnosis and accurately directed treatment may depend on the results of bacteriological investigations.

The next specimen (Figure 3a) was allowed to dry. There is no way that the pathologist can redeem a piece of tissue that has been mishandled in this way. Interestingly enough, a re-biopsy of this patient's vulval lesion, properly handled, disclosed carcinoma *in situ* (Figure 3b). It is the physician's responsibility to see that tissues are properly handled.

I'd like to show you one example of a proper biopsy taken from the rectum (Figure 4). Several fragments of tissue were received and processed. In one of these fragments, beneath intact mucosa and deep to the muscularis mucosae, are several little nests of tumor cells. This specimen enabled the pathologist to reach a diagnosis that might easily have been missed had the biopsy been less complete. This was a recurrent carcinoma that didn't show ulceration on the surface.

The last specimen that I would like to demonstrate was supplied by Dr. R. E. Johnson and deals with the aspiration biopsy. The patient from whom this tissue was removed gave a history of removal of a

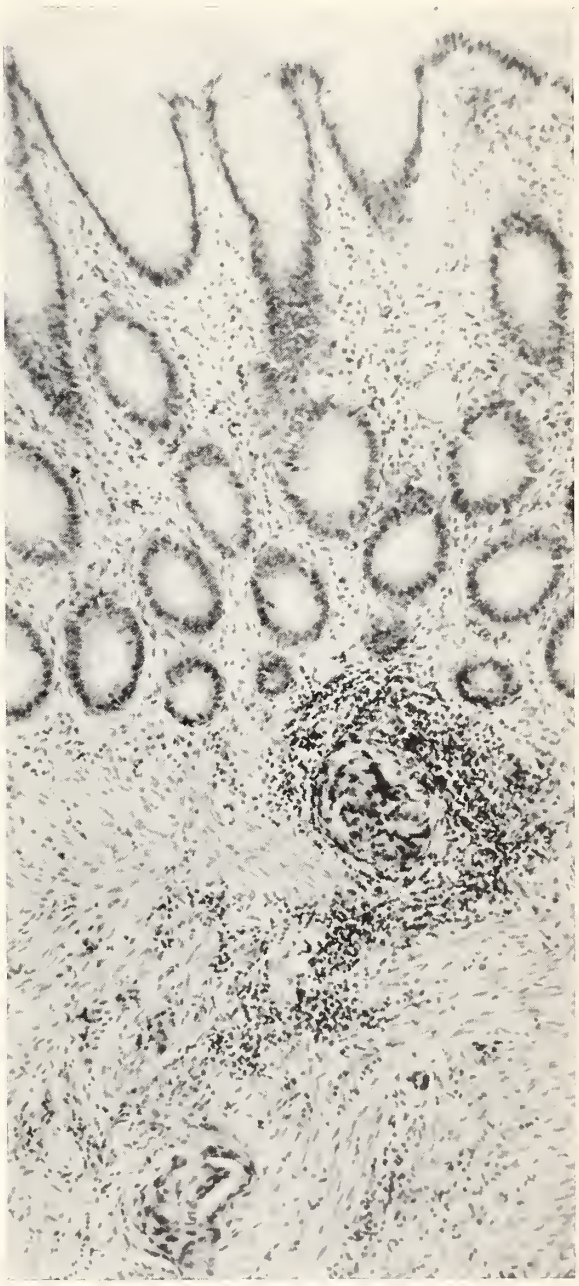


Figure 4. Rectal biopsy showing deeply situated nests of tumor cells beneath intact mucosa (mag. x100).

mole from the temporal area one year earlier. The mole had been treated with cautery, and no tissue was examined by a pathologist. A recurrent, rapidly enlarging nodule developed in the preauricular area on that side. Aspiration biopsy of this mass disclosed melanoma. Radical resection of the area followed a few days later, and a microscopic section shows extensive seeding of tumor cells along the biopsy needle tract (Figure 5). One cannot needle malignant tumors with impunity. If aspiration biopsy is done on a tumor that proves to be malignant, the entire needle tract must subsequently be removed, as was done in this case.



Figure 5. Depths of needle tract showing melanoma cells disseminated in subcutaneous fat (mag. x100).

Dr. Helwig: Dr. Johnson, do you care to comment?

Dr. Johnson: If aspiration biopsy is contemplated, the anticipated information should be of specific importance to the individual patient concerned. Otherwise, he should not be subjected to the risk of implantation of tumor by means of that aspiration. Secondly, we attempt to minimize the risk in operable cases by placing the needle tract in such a position that the subsequent surgical procedure can remove it.

The reason we aspirated a melanoma—some people would argue that one should never tamper with a melanoma in that way—was this. About one month before this 40-year-old man came to us, he developed a small nodule in the preauricular area on the right. The examination showed some little spots of pigment around the periphery of a flat achromic scar in the temporal region and a 3 cm. nodule in the preauricular area. We were suspicious, of course, that we were dealing with a melanoma, but because the situation called for wide excision of the skin of the face, total parotidectomy, and a radical neck dissection, including loss of the facial nerve, we felt that we should have histologic proof that it was melanoma and not some relatively benign lesion. So we did the aspiration biopsy, putting the needle tract in a position so that it was included in the subsequent radical dissection in continuity.

Dr. Helwig: Thank you, Dr. Johnson. I would like to add a note to what Dr. Robinson said about total excision biopsy. By removing for biopsy entire small skin lesions of less than 2 cm. in diameter, the physician will, in many instances, cure the patient. I would also like to emphasize the limited accuracy of the clinical diagnosis of certain types of skin lesions; xanthomas, seborrheic keratoses, histiocytomas, papillary pigmented basal cell epitheliomas, and others are often difficult to distinguish clinically. One dermatologist of wide experience has told me that his clinical impressions of this type of lesion are wrong in 20 per cent of instances. The average observer can't hope to do as well, and since these lesions can be eradicated by biopsy, this would appear to be the proper way to manage them.

Dr. Pollak, would you like to say a few words about the cervical smear?

Dr. Pollak: Cytologic diagnosis is a valuable technic in an office practice, provided the smears are taken properly. It is my personal opinion that a cervical smear should be a part of the routine pelvic examination. The making of the cytologic smear should be done by the physician; it should not be delegated to a technician. The smear should be made at the beginning of the examination, before lubricants have been introduced. Furthermore, the patient should

not have douched or bathed just before the specimen is acquired. I much prefer cervical smears to vaginal smears since I find that they yield more information.

Dr. Helwig: I would add that any mucus found in the cervical canal should be made a part of the smear; it often contains trapped cells. Always fix smears immediately, before they dry, in equal parts of alcohol and ether. Slides should remain in the fixative for 30 to 60 minutes and can be stored in this solution for weeks. A clean slide coated with glycerin will serve to cover the smear for mailing. Mailed slides should be carefully protected; the usual slide container too often proves inadequate.

Dr. Tice: One point that hasn't been mentioned is the medicolegal aspect of the biopsy. In treating cancer, radiologists often have to push treatment to the point where rather extensive damage occurs in tissues adjacent to the tumor. It is always a degree of satisfaction to have the pathologist's diagnosis of cancer as a basis for extensive therapy.

Dr. Robinson: I would like to make just one more point clear. As clinicians, we work with the pathologist in the interest of the patient. We are not antagonists of the pathologist. In the course of our examination of the patient we are likely to form a definite clinical impression concerning the patient's disease. When the pathologist doesn't confirm our impression we should get together with him and talk it over, and perhaps in conference we can arrive at a proper conclusion. Repeated biopsies are sometimes necessary to reach the correct diagnosis. One shouldn't relinquish clinical impressions too easily. The pathologist is removed from the patient; he often doesn't see the entire picture. Therefore, when he returns a negative report or one that is contrary to a strong clinical opinion, a discussion of the matter should follow. In a fair proportion of cases the diagnosis of the clinician will ultimately be confirmed.

BIBLIOGRAPHY

1. Neuberger, M., and Pagel, J.: Handbuch der geschichte der medizin, Jena, Gustav Fischer, 1905, p. 973.
2. Sternberg, S. S.: Pathology of juvenile nasopharyngeal angiofibroma—a lesion of adolescent males, Cancer 7:15-28, 1954.
3. Lattes, R., and Waltner, J. G.: Non-chromaffin paraganglioma of the middle ear, Cancer 2:447-468, 1949.

Statistics show that in 1927 the average cost of a case of lobar pneumonia was $1,000 and included the loss of at least five or six weeks of work. The chance for survival was only 3 to 1. In 1953, an average case of lobar pneumonia involved the loss of less than two weeks and was treated with antibiotics that cost from $15 to $30. The chance of survival was 25 to 1.—*Graydon L. Walker, Parke, Davis and Company.*

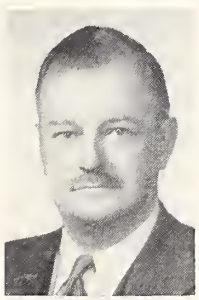
Mid-West Cancer Conference

The seventh annual Mid-West Cancer Conference, sponsored by the Kansas Division of the American Cancer Society and the Committee on Control of Cancer of The Kansas Medical Society, will be held at the Broadview Hotel, Wichita, on Thursday and Friday, March 24 and 25, 1955.

Committees planning the event have been working for months to arrange a program of interest to every physician in the state. Guest speakers who are widely known in the various specialties of medicine will take part in the session. There will be one big social event, a banquet for physicians and their wives, on Thursday evening.



Dr. Robert Dulaney Moreton, associated with the Tom B. Bond Radiological Group, Fort Worth, will be one of the speakers. Dr. Moreton is clinical assistant professor of radiology at the University of Texas, Southwestern Medical School. He is a fellow of the American College of Radiology and is a member of the Radiological Society of North America, Roentgen Ray Society, and Rocky Mountain Radiological Society. In 1949 he received two awards, one of which was a gold medal from the American Medical Association for an exhibit he had prepared and the other from the Association of Military Surgeons.



Also taking part in the program will be Dr. C. C. Little of Bar Harbor, Maine, who received his Sc.D. degree from Harvard University in 1914. Dr. Little has been active in cancer work for many years and in 1950 was winner of the American Cancer Society award for "Distinguished Service to Cancer Control." He is the author of a book, *Civilization Against Cancer,* published in 1939 and is a fellow of the National Academy of Sciences, New York Academy of Medicine, and National Institute of Social Sciences.

Biographical material about the other speakers will be published in subsequent issues of the JOURNAL. Names of the speakers are as follows: Dr. Richard Chamberlin, Philadelphia, radiologist; Dr. Elson B. Helwig, Washington, D.C., pathologist; Dr. John R. McDonald, Rochester, Minnesota, pathologist; Dr. J. Herbert Nagler, Philadelphia, oncologist; Dr. George T. Pack, New York, surgeon, and Dr. Henry K. Ransom, Ann Arbor, Michigan, surgeon.

COUNTY SOCIETIES

The new offices of the Sedgwick County Medical Society, 1102 South Hillside Avenue, Wichita, were formally opened on Sunday, October 24.

A scientific meeting of the society was held on November 2. Speaker was Dr. S. Gilbert Blount, Jr., assistant professor of medicine, University of Colorado Medical Center, Denver, who spoke on "Pathophysiology and Treatment of Chronic Cor Pulmonale."

Members of the medical societies in the first district entertained their wives at a dinner meeting held at the Sabetha Country Club on October 26. Dr. Frederick E. Wrightman, Sabetha, councilor for the district, presided. Speakers were Dr. Murray C. Eddy, Hays, president of The Kansas Medical Society, and Mrs. E. R. Millis, Kansas City, president of the Auxiliary.

A joint meeting of the Crawford County Medical Society and its auxiliary was held at the Hotel Besse, Pittsburg, November 4. Mr. Carl Brown and Mr. Lee Haworth were speakers, outlining local plans for civil defense and the part assigned to members of the medical profession.

Dr. Mahlon H. Delp, chairman of the Department of Postgraduate Education at the University of Kansas School of Medicine, was speaker at a meeting held at the Marion City Building on November 4. Present were physicians from Marion, Harvey, and McPherson counties.

About 50 persons were present at a meeting at the Concordia Country Club on November 4 when the Cloud County Medical Society entertained a number of guests. At the program following a dinner, Dr. Murray C. Eddy of Hays and Dr. John L. Lattimore and Mr. Oliver E. Ebel of Topeka were speakers.

Dr. Veryl J. Elson and Dr. Jack G. Rowlett, Paola, discussed "Virus Infections" at the October meeting of the Miami County Society held at the Presbyterian Church in Paola.

A meeting of the Wyandotte County Society was held at the City-County Health Building, Kansas City, on November 16. Dr. Richard F. Schneider discussed "Psychiatric Problems in General Practice," and Mr. John B. Smith, superintendent of the Winfield State Training School, told of the facilities available at the school and the work it is accomplishing.

Dr. Don C. Campbell, of the Mayo Clinic, was guest speaker at the November 1 meeting of the Shawnee County Society at Topeka. His subject was "Some Problems Related to the Anemias."

The Osage County Medical Society met at the Hotel Coffee Shop, Osage City, on October 22. Wives of the members were guests at a dinner which preceded the program. Speakers were Dr. Don C. Wakeman and Dr. John E. Crary, Topeka, who discussed "Diabetic Acidosis and Coma."

Dr. Philip W. Morgan, Emporia, was guest speaker at a meeting of the Cowley County Society held on October 21.

Members of the Harvey County Society and their wives met on November 4 at the Hotel Ripley, Newton. Dr. George Westfall, Jr., Halstead, was speaker of the evening.

Dr. Thomas A. Weldon, Perry, was host to the Jefferson County Society at a meeting held on October 28. A film on mental disease was shown.

Dr. Robert A. Dobratz, Chetopa, was elected president of the Labette County Society for 1955 at a meeting held at the Parsons Country Club on November 10. Dr. Arthur P. Burgess, Oswego, was named vice-president, and Dr. I. J. Waxse, Oswego, became secretary.

Diabetes detection drives were sponsored by three county societies, Atchison, Lyon, and Pratt, during "Diabetes Week," November 14-20. In Atchison County, free tablets for testing were provided through local drug stores. In Lyon and Pratt counties, physicians examined specimens from grade school students and from adults who asked for the service.

A meeting of the Wilson County Society was held in Neodesha on November 10, and the following officers for 1955 were elected: president, Dr. John W. McGuire, Neodesha; vice-president, Dr. Hugh G.

Bayles, Fredonia, and secretary-treasurer, Dr. Charles E. Stevenson, Neodesha.

The Committee on Public Relations of the Wyandotte County Society, under the chairmanship of Dr. Tony Dillon, is participating in a program of the American Cancer Society. Volunteers are being trained to visit the homes of persons chronically ill with cancer. Four physicians, Dr. Galen M. Tice, Dr. P. E. Hiebert, Dr. C. C. Nesselrode, and Dr. Maurice Laing, were instructors at the first session on "The Physical Problems of the Cancer Patient."

ACTIVITIES OF MEMBERS

Dr. George L. Thorpe, Wichita, attended the 105th annual session of the Indiana State Medical Association in Indianapolis late in October. He participated in a panel on "Emergencies" on October 26 and spoke on "Modern Office Therapeutics" before the general practice section of the meeting on October 27.

Among the speakers at the Sixth Annual Kansas Conference on Family Life held at Wichita on October 23 were Dr. Warren F. Bernstorf, Winfield, and Dr. Franklin D. Murphy, Lawrence.

Dr. S. Albert Scimeca, who has been practicing in Wichita, has announced the opening of an office in St. John.

Dr. Henry Laurens, Jr., Salina, recently completed a short postgraduate course at Temple University School of Medicine, Philadelphia, on the subject of diagnosis and treatment of diseases of the esophagus.

Dr. Howard V. Bair, superintendent of the Parsons State Training School, attended the Mental Hospital Institute of the American Psychiatric Association at Minneapolis, Minnesota, recently. He was certified by the group as a competent mental hospital administrator and was the recipient of one of the Achievement Awards for 1954.

The life story of Dr. William G. Norman, Cherryvale, was published as a feature story in the October 31 issue of the *Independence Reporter*. Dr. Norman, who is 82 years of age, has been in practice for 56 years, 47 in Cherryvale.

The Sedgwick County Medical Society announces that three of its members have returned to civilian practice after serving in the armed forces, Dr. Earnest P. Carreau, Dr. Perry D. Petterson, and Dr. William C. Swisher. All practice in Wichita.

Dr. Charles C. Hawke, Winfield, was speaker at the October meeting of the Cowley County Medical Assistants' Society held at the Snyder Clinic, Winfield.

Dr. Robert L. Stevens and Dr. Mildred J. Stevens, Garnett, returned last month from a trip to Europe. They were among 600 physicians from 52 countries attending the Eighth General Assembly of the World Medical Association, held in Rome. Other highlights of their trip were attending receptions given by the mayor of Rome and the mayor of Naples and participating in a group audience with Pope Pius XII.

Dr. Junior D. Burger, who has been practicing in Nickerson for six years, has announced plans to move to Hutchinson around the first of the year to practice there.

Dr. Joseph C. Shaw, Topeka, is the author of a column on early transportation which was published as a "Horse and Buggy Days" feature in the *Topeka Daily Capital* on October 25.

Dr. Daniel S. Roccaforte, who has completed two years as a surgeon with the Air Force at Smoky Hill Air Force Base, Salina, was recently released from service. He has returned to his practice at Hanover. Dr. Jack A. Haley, who practiced in Hanover during Dr. Roccaforte's absence, will leave soon to begin a residency in surgery.

Dr. Wayne L. Fowler, Concordia, recently attended a course in "Current Concepts in Internal Medicine," offered by the Chicago Medical Society.

The American College of Surgeons, at its annual meeting in Atlantic City last month, inducted 1,100 new fellows. Kansans in the group are Dr. Don R. Abbuehl, Chanute; Dr. Ernest C. Brandsted, McPherson; Dr. Stanley R. Friesen and Dr. Creighton A. Hardin, Kansas City; Dr. James E. Hill, Arkansas City; Dr. Alfred H. Hinshaw and Dr. Jerome S. Menaker, Wichita; Dr. Gaylord E. Manahan, Lawrence, and Dr. James A. McClure, Topeka.

DEATH NOTICES

Benjamin Smith Morris, M.D.

Dr. B. S. Morris, 54, an active member of the Central Kansas Medical Society, died at a hospital in Kansas City on October 24. Although he had been in poor health for several years, he had continued to practice in Quinter until three weeks before his death.

Dr. Morris, a graduate of the University of Kansas School of Medicine in 1925, practiced first in Morland. He moved to Quinter in 1927 and continued to practice there except for a short time in 1938 when he served in a government position in Missouri and Texas.

Joseph Fowler, M.D.

Dr. Joseph Fowler, 72, a Kansas physician since his graduation from the University Medical College of Kansas City in 1907, died at his home in Osawatomie, October 25. He was an active member of the Miami County Medical Society. Dr. Fowler practiced first in Paola, leaving there in 1916 to move to Fontana. Ten years later he went to Osawatomie and continued to practice there until his death.

George Wightman Kirby, M.D.

A physician who had practiced medicine in Wichita since 1919, Dr. George W. Kirby, 73, died at a hospital there on October 27 after an illness of three weeks. He was graduated from Rush Medical College, Chicago, in 1905 and began practice in Millersburg, Indiana. During World War I he served with the Army in an overseas surgical unit. He was an active member of the Sedgwick County Medical Society.

Arthur Edgar Bence, M.D.

Dr. A. E. Bence, 65, an active member of the Sedgwick County Society, died at his home in Wichita November 19 after a short illness. Dr. Bence, who specialized in orthopedics, had been practicing in Wichita since 1926. He was a graduate of the State University of Iowa College of Medicine with the class of 1921. He was a fellow of the American Academy of Orthopedic Surgeons.

ANNOUNCEMENTS

A six-day postgraduate course entitled "General Practice Review" will be offered at the University of Colorado Medical Center during the week of January 17-22, 1955. A separate area of practice will be presented each day as follows: Monday, medicine; Tuesday, pediatrics; Wednesday, surgery; Thursday, anesthesiology; Friday, obstetrics and gynecology, and Saturday, trauma.

Hotel reservations may be made through the office of Graduate and Postgraduate Medical Education, University of Colorado Medical Center, 4200 East Ninth Avenue, Denver 20, Colorado, until December 20. That office will also supply complete information and programs.

The International Academy of Proctology announces its annual cash prize and certificate of merit award contest for the best unpublished essay on proctology or allied subjects. First prize will be $100. Entries, limited to 5,000 words, must be received no later than February 1, 1955, at the offices of the Academy, 147 Sanford Avenue, Flushing 55, New York.

The Institute of Industrial Health of the University of Cincinnati is now accepting applications for a limited number of fellowships in industrial medicine. Any registered physician who is a graduate of a Class A medical school and who has completed at least two years of training in a hospital accredited by the A.M.A. may apply.

Stipends vary in accordance with the marital status of the individual, from $3,000 to $3,600 the first year to $3,400 to $4,000 in the second year. In the third year the candidate will be compensated for his service by the industry in which he is completing his training.

Additional information may be secured from the Institute of Industrial Health, College of Medicine, Eden and Bethesda, Cincinnati 19, Ohio.

A competitive examination for appointment of medical officers to the regular corps of the United States Public Health Service will be held in various places throughout the country on February 15, 16, and 17, 1955. Entrance pay for an assistant surgeon with dependents is $6,017 per annum; for senior assistant surgeon with dependents, $6,918. Application forms may be secured from the Chief, Division of Personnel, Public Health Service, Department of Health, Education, and Welfare, Washington 25, D.C. Applications must be filed before January 13, 1955.

Parkinson's Disease

Glen G. Halliday, M.D.

Portland, Oregon

In 1817, with a certain sense of apology, James Parkinson submitted "An Essay on the Shaking Palsy"[1] for publication. In its preface he says . . . "some conciliatory explanation should be offered for the present publication: in which, it is acknowledged, that mere conjecture takes the place of experiment; and that analogy is the substitute for anatomical examination, the only sure foundation for pathological knowledge. To delay . . . publication did not, indeed, appear to be warrantable. The disease had escaped particular notice; and the task of ascertaining its nature and cause by anatomical investigation, did not seem likely to be taken up by those who, from their abilities and opportunities, were most likely to accomplish it. That these friends to humanity and medical science, who already unveiled to us many of the morbid processes by which health and life is abridged, might be excited to extend their researches to this malady, was much desired; and it was hoped, that this might be procured by the publication of these remarks."

Historically, Parkinson credits Galen with having written of the disease, later Sylvius de la Boe, Juncker, and Cullen. To Parkinson goes first credit for using the term paralysis agitans, and his description of the syndrome is classically as follows: "Involuntary tremulous motion, with lessened muscular power, in parts not in action and even when supported; with a propensity to bend the trunk forwards, and to pass from a walking to a running pace; the senses and intellects being uninjured."[1]

Parkinson next discussed the natural history and course of this disease in detail. He concluded the first chapter by presenting six case histories. One of these (Case VI) is of special import. This patient was rendered hemiplegic by a cerebral vascular accident, and it was remarked . . . "neither the arm nor the leg of the paralytic side was in the least affected with the tremulous agitation; but as their paralyzed state was removed, the shaking returned." It was this case that later, perhaps, suggested to the neurological surgeon that he might have something to offer to certain patients.

In Chapter III, he gave a differential diagnosis which included palsy due to compression of the brain, alcoholic overindulgence, tea and coffee overindulgence, certain senile tremors, and anomalous cases of convulsive affections. It was his opinion that the pathological lesion was in the cervical portion of the spinal cord, later extended to the medulla oblongata, and that the signs and symptoms observed were due to interruption and irregularity in the direction of the nervous flow. Parkinson was of the opinion that treatment should consist of phlebotomy from the upper part of the neck, followed by application of vesicatories, and appropriate use of a substance to obtain a purulent discharge.

Prior to the appearance of epidemic encephalitis in 1917, Parkinson's disease was by and large a disease of later years, seldom occurring before 40 and usually making its initial appearance between 50 and 60. Cases seen early in life were of enough interest to warrant special reports, and the name of juvenile paralysis agitans was used. The following classification was compiled from a number of sources.

1. Syphilitic—Neill[3] recently reported a case of congenital syphilis in which several typical features of Parkinsonism were seen. Guillain[4] in 1935 and Wilson[5] in 1940 reported cases due to acquired syphilis. The signs may appear soon after the original infection or may be delayed for many years. The syndrome has also been seen in combination with tabes dorsalis and luetic paresis.[3] In Neill's case, improvement was witnessed following treatment with penicillin.

2. Toxic effects of carbon monoxide, manganese, cyanide, and carbon disulfide are all reported as causes of the disease.[6, 7, 8]

3. As a sequel to epidemic encephalitis (encephalitis lethargica, Type A encephalitis, von Economo's encephalitis)—Post-encephalitic Parkinsonism first attracted attention during the epidemic of 1917-1925. Boyd[9] states that around 20 per cent of patients with this type of encephalitis will subsequently exhibit one or more of the features of Parkinsonism. These cases are usually younger, and rigidity is often more prominent than tremor.

4. Arteriosclerotic changes can cause Parkinsonism, and this condition is a distinct and increasingly more common one characterized by an abrupt onset in later life. The tremor is coarse and invariably bilateral. There are often coincident findings which suggest diffuse cerebral arteriopathy (atherosclerosis)

This is one of 11 theses, written by fourth year students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group judged to be the best by the faculty at the school.

Thesis written while the author was a fourth year student. Dr. Halliday is now interning at Good Samaritan Hospital, Portland, Oregon.

such as dementia or bulbar dysfunction.[6]

5. "Idiopathic" degenerative paralysis agitans is a progressive degenerative disease of late middle life, characterized classically by tremor, with rigidity being a less prominent feature.

6. Due to acute hemorrhage into the basal ganglia.[8]

7. Due to neoplasms.[8]

8. Due to trauma.[7]

The understanding of the clinical manifestations of Parkinson's disease and any discussion of the pathological lesions is dependent upon a knowledge of the extrapyramidal motor system. This is defined by DeJong[7] as the most highly developed portion of "the old motor system." It is closely allied with the centers of motor integration in the brain stem concerned with regulation of tone and primitive postural and righting reflexes. It is also connected with those portions of the cerebral cortex which have to do with motor control. The nuclear centers of the extrapyramidal system, generally speaking, are situated in the basal ganglia, and Parkinson's disease is frequently referred to as a disease of the basal ganglia or of the striatum. Stated dogmatically, these centers are the caudate and lenticular, the latter being composed of the putamen and globus pallidus.

There are other structures which are functionally and clinically related to the extrapyramidal system. These are the subthalamic nucleus or corpus Luysi, red nucleus, and substantia nigra. In addition, there are extra pyramidal portions of the cerebral cortex—that portion anterior to the premotor area of the frontal cortex and another area in the parietal cortex. The basal ganglia and the related structures constitute an important efferent system. The parts of the cortex mentioned above discharge to the midbrain nuclei, the red nucleus and substantia nigra in part, and these latter structures in turn discharge to the brain stem and the spinal cord.

Although dominated by the cerebral cortex, the "old motor system" has a steadying influence on volitional movements and may be utilized for the initiation and production of many more or less automatic movements. It is these habitual movements of daily life that are most impaired in diseases of the basal ganglia.

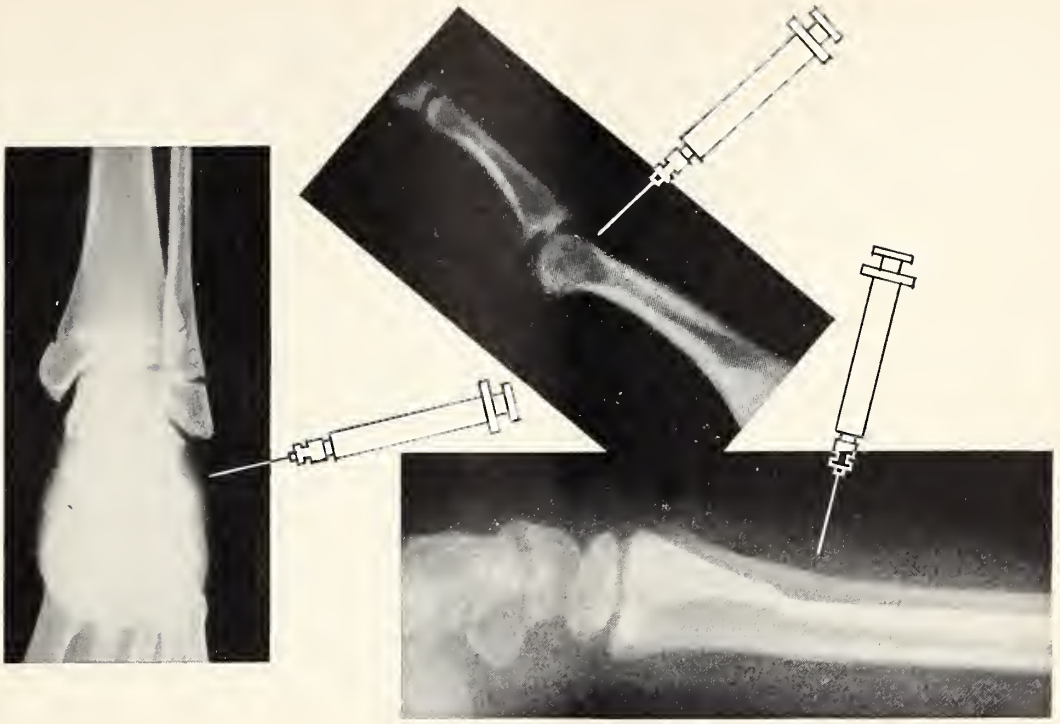
It is generally agreed now that the pathological lesions of Parkinson's disease are in the extrapyramidal motor system. Gower (1888), Omerod (1890), Anton (1895), and Jelgersma (1909) noted changes in the basal ganglia in chorea, athetosis, and paralysis agitans, but the first adequate descriptions of the relationship of neurologic symptoms were made in 1911 and 1912 by Alzheimer, Vogt, Wilson, and Lewy.[7]

Hunt (1917)[10] demonstrated atrophy of the ganglion cells of the globus pallidus in cases of typical Parkinson's disease. Jacob (1925) showed degeneration also of the substantia nigra.[10] Boyd[9] states that the cause of the degeneration is not known and suggests it might be in the form of a senile atrophy, also saying that lesions in post-encephalitic Parkinsonism were found in both the red nucleus and the substantia nigra. Oseki[10] (1924) examined brains from senile patients who had not had Parkinson's disease and saw changes identical with those ascribed to this disease. Oliver[10] mentions that cases have been reported in which the tremor and rigidity were unilateral and the microscopic degenerative changes bilateral.

In general it may be said that disease of the striatum is characterized by some positive and some negative signs and symptoms: (1) general increase of muscle tonus, that is rigidity; (2) abnormal involuntary movements, that is tremor; (3) poverty of voluntary, especially spontaneous movements, namely hypokinesia; (4) impairment of automatic associated movements; (5) absence of "true" paralysis, namely absence of signs of involvement of pyramidal tracts; (6) absence of sensory disturbances.

The signs and symptoms of Parkinson's disease and the general order of their appearance are so aptly described in the original essay[1] that the following is given verbatim: "So slight and nearly imperceptible are the first inroads of this malady, and so extremely slow is its progress, that it rarely happens that the patient can form any recollection of the precise period of its commencement. The first symptoms perceived are, a slight sense of weakness, with a proneness to trembling in some particular part; sometimes in the head, but most commonly in one of the hands or arms. These symptoms gradually increase in the part first affected; and at an uncertain period, but seldom in less than 12 months or more, the morbid influence is felt in some other part. Thus assuming one of the hands and arms to be first attacked, the other, at this period becomes similarly affected.

"After a few more months the patient is found to be less strict than usual in preserving an upright posture; this being most observable whilst walking, but sometimes whilst sitting or standing. Sometime after the appearance of this symptom, and during its slow increase, one of the legs is discovered slightly to tremble, and is also found to suffer fatigue sooner than the leg of the other side; and in a few months this limb becomes agitated by similar tremblings, and suffers a similar loss of power. As time and disease proceed . . . difficulties increase; writing can now be hardly at all accomplished; and reading, from the tremulous motion, accomplished with some difficulty. At this period . . . the patient has recourse to walking, a mode of exercise to which the sufferers from



Use of Alidase® in Closed Wounds: Contusions, Sprains, Dislocations, Simple Fractures

In traumatic surgery[1] where "definitive treatment . . . is often delayed while the surgeon waits for nature to dispose of hematoma and oedema" Alidase is an efficient means[1, 2] of accelerating dispersion of accumulated fluids.

Swenson[2] has described his highly successful results with Alidase in various types of closed wounds. He summarized them as follows:

To remove local fluid accumulations in contusions or bruises, "The usual dose, 500 viscosity units Alidase® mixed in a small amount of normal saline, is injected into the localized fluid. Mixing the hyaluronidase in 1 per cent procaine solution will also produce local vasodilatation, relief of local pain and more rapid absorption of the fluid mass. This method can also be applied to traumatized bursae or synovial spaces which do not respond to repeated aspirations."

The point of maximal pain is infiltrated with 10 cc. of a 1 per cent procaine solution to which 500 viscosity units of Alidase have been added. With this simple technic, a high percentage of successful results has been obtained.

Alidase may be used to advantage to produce more rapidly a short-acting, complete block anesthesia and to facilitate reduction in subluxation or complete dislocations of the interphalangeal joints. When anesthesia is required for fracture reduction, local block anesthesia can be simplified by adding Alidase to the anesthetic solution. Alidase also tends to decrease local edema and hematoma formation.

Fluids administered with Alidase are rapidly absorbed from subcutaneous tissue. The simplicity of hypodermoclysis avoids the cumbersome arm board, permits convenient administration with little or no pain or swelling, is vein-sparing and saves nursing time in such conditions as burns, postoperative states, toxemias and parenteral alimentation.

Alidase (brand of hyaluronidase) is supplied in serum-type ampuls of 500 viscosity units. It is accepted by the Council on Pharmacy and Chemistry of the American Medical Association. G. D. Searle & Co., Research in the Service of Medicine.

---

1. MacAusland, W. R., Jr.; Gartland, J. J., and Hallock, H.: The Use of Hyaluronidase in Orthopaedic Surgery, J. Bone & Joint Surg. *35-A*:604 (July) 1953.
2. Swenson, S. A., Jr.: Minor Surgical Aspects of Closed Wounds, Am. J. Surg. *87*:384 (March) 1954.

this malady are in general partial; owing to their attention being thereby somewhat diverted from their unpleasant feelings . . . but as the malady proceeds, even this temporary mitigation of suffering . . . is denied. The propensity to lean forward becomes invincible, and the patient is thereby forced to step on the toes and fore part of the feet, whilst the upper part of the body is thrown forward as to render it difficult to avoid falling on the face . . . being at the same time, irresistibly impelled to take much quicker and shorter steps, and thereby to adopt unwillingly a running pace.

"In this stage . . . the tremulous motions of the limbs occur during sleep, and augment until they awaken the patient. . . . As the disease proceeds toward its last stage, the trunk is almost permanently bowed, the muscular power is more decidedly diminished, and the tremulous agitation becomes violent. His words are now scarcely intelligible . . . so much are the actions of the muscles of tongue and pharynx impeded . . . another very unpleasant circumstance occurs; the saliva fails of being directed to the back part of the fauces, and hence is continually draining from the mouth . . . the tremulous agitation becomes more vehement. It now seldom leaves him for a moment; . . . the power of articulation is lost. The urine and faeces are passed involuntarily; and at the last, constant sleeplessness, with slight delirium, and other marks of extreme exhaustion, announce the wished-for release."

Perhaps the most significant omission from this masterful description is the waxy, masklike, rigid facies so often seen. To sum up, the salient signs and symptoms are (1) coarse, rhythmic, pill-rolling tremor, usually one of rest, sometimes extending to the tongue and head, (2) plastic muscular rigidity, (3) masklike facies, (4) ante-flexed posture, (5) loss of associated arm movements when walking, and (6) hesitant initiation of walking, with festination, propulsion; retropulsion, or lateropulsion and acceleration. The diagnosis of a fully developed case of Parkinson's disease offers no difficulty.

However, the early diagnosis of this condition is not so easy. This is discussed in detail in a recent article by Wartenberg.[11]

The course of the disease is slow, often lasting 5, 10, or 15 years. Ultimately every patient becomes bedridden and has to be fed and nursed. The prognosis is hopeless and, while the disease itself does not kill, there is no chance of recovery.

The treatment of Parkinson's disease is essentially one or a combination of the following: medical, surgical, by physiotherapy, or by psychotherapy.

The ever-increasing number of drugs introduced and extolled is sufficient indication that none is entirely satisfactory. Certainly there is nothing to even approach the efficacy of cyanocobalamine for pernicious anemia or insulin for diabetes mellitus. Some of the drugs available will give relief and bring about improvement in some patients and not in others; certain of symptoms may be improved and others not. Indeed, there not only are innumerable articles dealing with treatment, there are even articles[12] discussing the hazards and common fallacies present in evaluation of therapeutic results. Extreme divergence of opinion has been characteristic of therapeutic claims since the original paper.

As was mentioned previously, Parkinson used phlebotomy and induced infection for treatment.[1] Charcot suggested horseback riding and eventually devised a contraption in which his patients would sit and be shaken for prescribed periods of time. In 1874,[15] Charcot introduced belladonna therapy in this syndrome. In 1882,[13] Gnauck discovered the effectiveness of hyoscine (scopolamine) in palliation of the symptoms of Parkinson's disease. This opened the present era of medical therapy. Later, other alkaloids of the solanaceous group were noted to have similar effects. These were atropine, hyoscyamine, extracts of the stromonium leaf, and extracts of other plants containing the mydriatic alkaloids.

Through the years, many drugs for both oral or parenteral administration have been tried and have fallen into disuse, such medicaments as quinine, neostigmine bromide, iodides, magnesium sulfate, amphetamine, snake venom, salicylates, and phenobarbital. Vitamins of all types and combinations have had their periods of enthusiastic acceptance but failed to stand the test of time.[16]

Therapy must be individualized, and often trial and error with drug combinations will discover what is most successful for a certain patient. The effective drugs fall essentially into three groups. These are the belladonna or solanaceous drugs, the antispasmodics, and the antihistaminics.

A. THE BELLADONNA GROUP

1. Atropine sulfate in 0.5 per cent solution. This is said to be of special value in the younger age groups and in post-encephalitic cases.[14] The recommended dosage is 10 to 30 drops three times a day. A recent article[17] suggests giving initial doses of three drops at intervals of at least six hours because of slow rate of excretion. The dose is then increased one drop three times a day every three days until a dosage of ten drops three times a day is reached. Excessive dosage may produce vomiting, mental confusion, dizziness, and hallucinations.[17] Other undesirable symptoms are blurring of vision, dryness of skin and mucous membranes, somnolence, and tachycardia.[18]

2. Hyoscine hydrobromide is still one of the most

one of the 44 uses for short-acting **'Nembutal'**

In a matter of moments, her nerves will be calmed. Her anxiety will be alleviated. And her tensions will slide into somnolence.

Short-acting NEMBUTAL (Pentobarbital, Abbott) can produce any desired degree of cerebral depression—from mild sedation to deep hypnosis.

The dosage required is small—only about one-half that of many other barbiturates.

Hence, there's less drug to be inactivated, shorter duration of effect, wide margin of safety and little tendency toward morning-after hangover.

In equal oral doses, no other barbiturate combines quicker, briefer, more profound effect.

Good reasons why the number of prescriptions for short-acting NEMBUTAL continues to grow—after 24 years' use in more than 44 clinical conditions.

Abbott



For mild sedation try the 50-mg. (¾-gr.) NEMBUTAL Sodium capsule.

412207

useful preparations, especially in reducing tremor and excitement.[14] The dose is 0.25 to 0.6 milligrams two to three times a day. Signs of toxicity are generally the same as those given for atropine.

3. Tincture of stramonium has been employed for years to control rigidity and tremor. The effect upon rigidity is less than that obtained with some of the newer preparations as a general rule. The tincture is prescribed in doses of 10 to 20 drops three times a day or, in the tablets, in two-grain dosage three times a day.[14] According to some,[17] this drug is easily tolerated by the elderly.

4. Rabellon (Sharpe and Dohme) is a compound of belladonna alkaloids containing 0.5 mg. of atropine and hyoscyamine with traces of scopolamine. It is given in one-half, one-fourth, or full tablets two, three, or four times a day. Although a good muscle relaxant, Rabellon has little action against tremor.[17]

5. Vinobel, which is dispensed in tablets containing 0.4 mg. of the active alkaloids for elderly patients and 0.8 mg. for young persons, is an effective antispasmodic.[14, 17] Side reactions are those of the other belladonna products.

6. Bellabulgara is prepared in tablets containing 0.45 mg. of the alkaloids atropine and hyoscyamine and a trace of scopolamine.[17] In the late 1930's and early 1940's, this preparation was in particular vogue.[15] It was thought at this time that it possessed some particularly efficacious properties. It is administered in one-half or full tablet doses three times a day.

B. THE SYNTHETIC ANTISPASMODIC DRUGS

1. Artane (Trihexiphenidyl) is manufactured by Lederle Company. It is perhaps the most important recent addition to the drugs used for Parkinson's disease. Pharmacologically, Artane exerts both atropine-like and antihistaminic effects.[19, 22] On isolated intestine loops, its relaxant action is half that of atropine.[19] It is mildly excitant to the central nervous system but much less so than atropine.[19] It is one per cent as potent a vagus inhibitor as atropine.[22] Salivation is but slightly inhibited.[19]

A recent article[17] stated that Artane is recommended as the basic drug in treatment of all new cases. It may be used at all age levels.[17] Its chief advantage over the traditional belladonna drugs is in the wider margin of safety existing between the effective and toxic doses. Another advantage is that Artane doses need not be continuously increased in order to maintain its action.[19] It may be safely used in patients with cardiac disease, hypertension, hepatic or renal disease.[14, 22] It had no ill effects upon blood pressure, respiration, blood count, renal or hepatic function, sugar metabolism, growth, and reproduction in laboratory animals over a six-months period.[22]

The first six reports[20] in the literature appeared in 1949 and 1950. Each was a large series, and combined results listed improvement in 76 per cent of cases. They reported Artane to be most effective in controlling rigidity and spasticity with 10 per cent incidence of mild side reactions and 4 per cent incidence of severe side reactions. The patient should be started on 1 mg. each day. This is increased 1 or 2 mg. each day to a total of 6 to 10 mg., which is divided into three or four doses.

2. Curare (placed in this group for convenience) was tried about 1927 and abandoned.[21]

3. Diparcol is a synthetic antispasmodic developed in France. Results with it were not too satisfactory. Its manufacture has been discontinued because of its toxic manifestations and occasional cases of agranulocytosis.[6]

4. Tolserol (Myanesin, Mephanesin) is given first as one 250 mg. tablet three times a day and rapidly increased to three, four, or five tablets three or four times a day.[22] One group[22] used Tolserol in 22 patients and had to discontinue it in all but one because of nausea, precordial discomfort, and pronounced increase of tremor.

5. Panparnit (Carmiphen hydrochloride) was developed in Switzerland. Results have not been good, and a high incidence of severe toxic effects is reported.[14, 22]

6. Parsidol comes in 50 mg. tablets. It is administered in one-half tablet doses four times a day to elderly patients and full tablets four times a day to younger persons. Results so far have been poor.

C. THE ANTIHISTAMINICS

1. Benadryl. It was appreciated by early investigators that the anticholinergic activity of antihistamine substances might be of value in the therapy of Parkinson's disease. The use of such substances began with the treatment of four patients as a part of a routine screening of Benadryl in many diseases.[23]

Budnitz,[24] in 1948, released a preliminary report concerning eight patients suffering with Parkinson's disease who were treated with Benadryl. He observed that "all of them in the arteriosclerotic group were treated with Benadryl over a period varying from 3 to 14 months. All patients noted considerable improvement in the symptoms as long as the drug was administered. Four of the patients treated continued to use parasympathetic inhibitory drugs of the atropine series, and there seemed to be a synergistic action between these drugs and Benadryl."

Moore[25] reported a series of 52 patients using Benadryl and Benadryl and hyoscine. He stated that

WHEN SYMPTOMS ARE DISTRESSING BUT DISGUISED . . .

"It is strange," Malleson says, "how little clinical recognition" has been given to the "negative behavior" or "endogenous misery" of the woman with endocrine imbalance. Largely accountable for this, of course, is the patient's own reluctance to discuss these symptoms with her physician until she actually suffers from some of the more obvious menopausal symptoms such as hot flushes. Even then she may become so accustomed to her change in feeling she can't remember what it's like to feel well.[1]

Changes in the mood pattern are just a few of the many distressing symptoms of declining ovarian function which are so often disguised because they do not always coincide with cessation of menstruation, and at times will occur long before, and even years after. Other good examples are insomnia, headache, easy fatigability, arthralgia — and understandably so, when one considers that the loss of ovarian hormone "withdraws one of the most important metabolic regulators of the organism."[2]

"Premarin" is a preparation of choice for the replacement of body estrogen. "Premarin" presents a *complete* equine estrogen-complex and all the components of this complex are meticulously preserved in their natural form. This largely explains why "Premarin" not only produces prompt symptomatic relief but also imparts an important "plus" — the distinctive "*sense of well-being*" that patients find so highly gratifying. These benefits of "Premarin" have made it a natural estrogen widely prescribed by physicians . . . and often preferred by patients.

"PREMARIN"®

ACCEPTED COUNCIL ON PHARMACY AND CHEMISTRY AMERICAN MEDICAL ASSOCIATION

has no odor
. . . imparts no odor

Estrogenic Substances (water-soluble), also known as conjugated estrogens (equine), available in both tablet and liquid form

1. Malleson, J.: Lancet 2:158 (July 25) 1953. 2. Goldzieher, M. A., and Goldzieher, J. W.: Endocrine Treatment in General Practice, New York, Springer Publishing Company, Inc. 1953, p. 23.

NEW YORK, N. Y. Ayerst MONTREAL, CANADA

5409

his patients did not fare well on Benadryl alone but showed marked improvement on Benadryl plus a member of the atropine group. Twenty-five per cent of his patients had drowsiness which was eliminated when amphetamine was administered upon arising and at noon. This was the only distressing side effect noted.

At present, Benadryl is one of the most often used preparations. In a recent article,[17] it was stated that Benadryl controls tremor well, allays tension and excitement, and aids patients with insomnia. Side reactions of somnolence and fogginess may so disturb elderly patients as to limit use to bedtime. Usually 50 mg. doses are given two to four times daily. Bercel[26] states that of the various symptoms of Parkinsonism, subjective complaints due to vegetative nervous system dysfunction—muscle cramps, rigidity, diaphoresis, sialorrhea, asthenia, emotional incontinence, and tremor—were most favorably affected. He also stated that a six-weeks trial was necessary for evaluation.

2. Thephorin is perhaps the second antihistaminic which should be mentioned. Cohen and Criep[27] state that a few months before the first articles about Benadryl and Parkinsonism appeared, they had noticed considerable symptomatic improvement in a patient with Parkinson's disease who was receiving Thephorin for coexisting allergic rhinitis. They conducted a series using Benadryl, Pyribenzamine, and Thephorin. They stated that Thephorin resulted in the greatest degree of improvement with tremor, hypertonicity, and rigidity being most frequently benefited. The dosage recommended is 25 mg. two or three times a day.

3. Pyribenzamine has not been of value in treatment of Parkinson's disease.[13, 27]

4. Histadyl was reported to elicit poor results.[13]

Hand[14] states that Benadryl and Thephorin are the only antihistaminics of value in Parkinson's disease. These agents are perhaps synergistic with Artane; by the trial and error method, some combination of the three above discussed groups of drugs should be found which will afford a degree of palliation for the sufferer.

Parkinson's Case VI, in whom temporary unilateral cessation of symptoms was observed following a cerebral vascular accident, was evidence that destructive neurosurgical procedure might have a place in the treatment of selected cases.

A recent monograph by Oliver[10] discusses this aspect of treatment in adequate detail. Of slightly more than 1000 patients examined, 300 were admitted for further hospital study, and 93 eventually were selected for operation. He noted that no abnormalities in spinal fluid examination were discovered. Also, the scalp leads from an electroencephalograph revealed no unusual characteristics. Fifty cases were studied by pneumoencephalograms, and no convincing abnormality was observed.

Numerous operations have been proposed. Essentially all are alike in that the corticospinal tracts are sectioned at some point in their descent. The operations differ as to the site selected, cortex or cervical cord, and the depth to which the scalpel is pushed. It would seem, in a certain small percentage of cases in which tremor is unilateral, that surgery might be of value. The vast majority of patients with Parkinson's disease will be in the older age groups. Special efforts should, therefore, be directed toward being as certain as possible that the patient can tolerate surgery.

Physiotherapy should be confined to increasing muscle tone rather than to providing momentary comfort to the patient.[17] It should consist of massage, stretching of muscles, and active exercise with special equipment.[17] Heat lamps, diathermy, and ultraviolet machines are of little value. Physiotherapy, if enthusiastically administered, will always produce some improvement. An occasional short course of active exercises from a physiotherapist with an energetic and optimistic approach may sometimes help these patients make full use of any persisting potential capacity for physical activity.[6]

As in the treatment of most diseases, psychotherapy is of value in Parkinson's disease. Psychotherapy is perhaps no more than a conscious effort to maintain a good doctor-patient relationship. It is that sort of common-sense encouragement and reassurance which every physician is called upon to provide every day.

Quite naturally, many with this disease tend to become sorry for themselves, and they need to be protected from the over-sympathy which they receive from their friends and families and to be encouraged to keep going as long as possible. They are also prone to consider their disability as more conspicuous than it really is. The most important factor is undoubtedly to try to maintain the patient's morale, and Parkinsonism is one of several incurable chronic diseases of the nervous system in which the most effective treatment is psychotherapy in its least specialized sense, administered consciously or unconsciously by an understanding practitioner.

Parkinson's disease, known for centuries but first described by James Parkinson in 1817, is a slowly progressive affection of the central nervous system. It can be caused by arteriosclerosis, encephalitis lethargica with an interval of several years before symptoms appear, syphilis, carbon monoxide, carbon disulfide, manganese, hemorrhage, trauma, or neoplasm, or it may be of the so-called idiopathic type. The signs and symptoms are discussed with an attempt to correlate the clinical findings with the altered physiology of the extrapyramidal system.

Karo

helps to support this dramatic growth!

. . . a carbohydrate of choice in milk modification for 3 generations

For the newborn

Karo Syrup is a milk additive that is hypoallergenic and bacteria-free. Since it is rich in easily digested dextrose, maltose and dextrins, it provides carbohydrates in directly assimilable form. This minimum demand on the digestive function is important during the first weeks. It makes possible a formula containing 15 calories per ounce even during the period when fat digestion is least efficient.

During the first months

When growth is most rapid, Karo helps to meet the accelerated nutritional demand. It offers in convenient, well tolerated form the carbohydrate additive which is usually prescribed, since milk alone provides just 28% of the optimum 60% carbohydrates. Karo Syrup is also readily available, inexpensive, a miscible liquid that is easy to use. Light and dark Karo are interchangeable in formulas—both yield 60 calories per tablespoon.

For the older infant

Karo eases the transition from formula to whole milk, from liquid to solid foods. The familiar taste of Karo makes whole milk more readily accepted, and many solid foods will be easily introduced into the diet if flavored with a little Karo Syrup. Rapidly assimilable carbohydrate is needed for the rapid metabolism of the small child. Since Karo is low in osmotic pressure, it is non-irritating. It also precludes fermentation because no excess of hydrolized sugars is formed.





Medical Division

CORN PRODUCTS REFINING COMPANY

17 Battery Place, New York 4, N. Y.

Although there is no cure for the disease, therapy can and should be actively and energetically undertaken by the physician. Therapy was discussed as medical, surgical, physiotherapeutic, and psychotherapeutic. Artane and Benadryl are probably the most efficacious drugs available. It was stressed that combination therapy be used until the most improvement is noted. An occasional patient may be a candidate for surgery, and the criteria for this are discussed. Physiotherapy is of limited value. Of perhaps the most value is the commonsense psychotherapy which every physician can employ if he will. The patient should be urged and encouraged to remain as active as his disabilities allow.

REFERENCES

1. Parkinson, J.: An Essay on the Shaking Palsy, London, Whittingham and Rowland, 1817. Facsimile reproduction, London, Sherwood, Neely, and Jones, 1925, and Chicago, American Medical Association, 1917.
2. Wechsler, I.: A Textbook of Clinical Neurology, ed. 7, Philadelphia, W. B. Saunders Company, 1952.
3. Neill, K.: An unusual case of syphilitic Parkinsonism, British Medical Journal 2:320 (August 8) 1953.
4. Guillain, G.: Progr. med., Paris, 50, 978, 1935.
5. Wilson, S. A. K.: Neurology, ed. 1, London, Arnold, 1940, vol. 1, p. 483.
6. Miller, H.: Parkinsonism and its management, Practitioner 171:1021 (July) 1953.
7. DeJong, R.: The Neurologic Examination, ed. 1, New York, Paul B. Hoeber, Inc., Medical Book Department of Harper and Brothers, 1950.
8. Wechsler, I.: A Textbook of Clinical Neurology, ed. 6, Philadelphia, W. B. Saunders Company, 1947.
9. Boyd, W.: A Textbook of Pathology, ed. 5. Philadelphia, Lea and Febiger, 1950.
10. Oliver, L.: Parkinson's Disease and its Surgical Treatment, London, H. K. Lewis and Company, Ltd., 1953.
11. Wartenberg, R.: Head dropping test, British Medical Journal 1:687 (March 29) 1952.
12. Vollmer, H.: Pitfalls in evaluating drug therapy in Parkinsonism, New York State Journal of Medicine 51:1933 (August 15) 1951.
13. Hand, B.: Anti-convulsant, anti-epileptic, anti-Parkinsonism Drugs, American Practitioner and Digest of Treatment 4:17 (January) 1953.
14. Gair, D., and Ducy, J.: Chemical structures of substances effective in treatment of Parkinsonism, Archives of Internal Medicine 85:284 (February) 1950.
15. O'Doherty, D., and Forster, F.: The use of henzotropine sulfonate in treatment of Parkinsonism, Medical Annals of the District of Columbia 22:221 (May) 1953
16. Effron, A., and Denker, P.: A clinical evaluation of certain antihistaminic and antispasmodic drugs in Parkinson's disease, Journal of the American Medical Association 144:5 (September 2) 1950.
17. Dorshay, L., and Zier, A.: The therapy of Parkinsonism, Medical Clinics of North America (September) 1953.
18. Yaskin, J.: Treatment of Parkinson's syndrome, Pennsylvania Medical Journal 54:1156 (December) 1951.
19. Canelis, M.; Farnel, F., and McGavack, T.: Clinical experiences in Parkinsonism with a new type of antispasmodic, American Journal of the Medical Sciences 218:635 (December) 1949.
20. Harris, T., and Torrens, J.: Use of artane in Parkinsonism, Texas State Journal of Medicine 46:514 (July) 1950.
21. Garland, H.: Parkinsonism, British Medical Journal 1:153 (January 19) 1952.
22. Dorshay, L., and Constable, K.: Newer drugs in the treatment of Parkinsonism, Neurology 1:68 (January-February) 1951.
23. McGavach, T.; Elias, H., and Boyd, L.: Some pharmacologic and clinical experiences with dimethyl-amino-ethyl benzhydryl ether hydrochloride. American Journal of the Medical Sciences 213:418 (April) 1947.
24. Burnitz, J.: The use of benadryl in Parkinson's disease, New England Journal of Medicine 238:874 (June 17) 1948.
25. Moore, M.: Benadryl in the treatment of Parkinson's disease, Neurology 1:123 (March-April) 1951.
26. Bercel, N.: Clinical evaluation of benadryl in Parkinsonism, American Practitioner and Digest of Treatment 2:1042 (December) 1951.
27. Cohen, S., and Criep, L.: Observations on the use of antihistaminics in Parkinson's disease (paralysis agitans), Journal of Nervous and Mental Disease 115:57 (January) 1952.

Good public relations begins in the office of each individual physician.

BOOK REVIEWS

A History of Medicine, Volumes 1 and 2. By Ralph H. Major. Published by Charles C Thomas, Springfield, Illinois. 1155 pages. Price $14.50.

In the preface Dr. Major defines the purpose and scope of his work. "This book is not written for the specialist in medical history, to whom most of the facts related are already well known. It is written primarily for the medical student and the medical practitioner, in an attempt to interest them more in the history of their own profession, which, despite attacks and abuse, despite quacks and charlatans, and in spite of its own mistakes, has proved itself more enduring than any of the civilizations which gave birth to it.

"To include in a history of medicine the names of all physicians who have made notable contributions to medicine would transform such a history into either an exhaustive reference book or a mere dictionary of medical names. To avoid this dilemma, I have attempted to write a continuous account of the stream of medical history, punctuated with the names of eminent physicians often accompanied by a brief biography."

A History of Medicine begins in antiquity and traces achievements of the various cultures and nations to the middle of the 20th century. It is a compelling narrative regularly enriched with an inspired selection of quotations from the period or the physician under discussion. And not least among its values are the more than 400 distinctive and informative illustrations, many being photographs taken by the author himself.

It is a readable book beautifully printed on fine paper. *A History of Medicine* will be of interest to the medical student and the medical practitioner. It will be a popular source reference in every library, but it has a wider potential influence than in professional circles.

The author uses the historian's dispassionate approach, but his subject is filled with the breathless suspense of discovery, the terror of plagues, and the comfort of continual progress. It is science, but the science of humanity. It is history, but more vital than the conquest of armies. It is fact, but the fact of living, personal experience.

So this two-volume record of medicine would be an ideal gift. It would be an inspiration to the young man and woman who dream of becoming physicians and a satisfaction to the inquiring layman. Perhaps no argument for medicine has ever approached the eloquence of its own dignified and continual achievement. Dr. Major has not only preserved this record in the 1155 pages of his book but

ANNUAL CLINICAL CONFERENCE

CHICAGO MEDICAL SOCIETY

March 1, 2, 3, 4, 1955

Palmer House, Chicago

DAILY HALF-HOUR LECTURES BY OUTSTANDING TEACHERS AND SPEAKERS on subjects of interest to both general practitioner and specialist.

PANELS ON TIMELY TOPICS.

MEDICAL COLOR TELECASTS.

TEACHING DEMONSTRATIONS.

SCIENTIFIC EXHIBITS worthy of real study and helpful and time-saving TECHNICAL EXHIBITS.

The CHICAGO MEDICAL SOCIETY ANNUAL CLINICAL CONFERENCE should be a MUST on the calendar of every physician. Plan now to attend and make your reservation at the Palmer House.

It's Convenient to Buy Kanox

Send your patients to our sickroom department for all their needs. You may rest assured they will receive good courteous service at a reasonable price.

Walkers	Sales	Canes & Crutches
Wheelchairs	&	Commodes
Beds	Rentals	Supply Items

We are now stocking many and varied items used in hospitals and doctor offices. Would you give us an opportunity to service your account? Every article sold with an unconditional guarantee of satisfaction. Why not try us?

KANSAS OXYGEN, Inc.

Hospital Dept.

Phone 2-8981 Hutchinson 329 W. 4th St.

he has succeeded in bringing people into reality and in reenacting events before our eyes as we read. —*O.E.E.*

Galen of Pergamon. By George Sarton. Published by University of Kansas Press, Lawrence, Kansas. 112 pages. Price $2.50

Galen of Pergamon is the third volume in the Logan Clendenning Lectures on the History and Philosophy of Medicine to be published by the University of Kansas Press.

This is the readable account of the life of the famous and influential physician and makes for excellent bedside reading for every physician. Most of the physicians in Kansas have visited the Library of the History of Medicine and know that the Logan Clendenning Library is one of the great libraries of medical history in the world. Those who were not fortunate enough to hear Dr. Sarton present the material covered in the book will welcome this fine volume.—*W.L.V.*

Manual of Clinical Mycology, Second Edition. By Norman F. Conant, David T. Smith, Roger D. Baker, Jasper L. Callaway, Donald S. Martin. Published by W. B. Saunders Company, Philadelphia. 456 pages, 202 figures. Price $6.50.

This manual contains a concise description of the several recognized mycotic diseases together with discussion of epidemiology, diagnostic methods, and treatment. The sections on laboratory diagnosis are sufficiently detailed to serve as a reference, and a brief chapter on general mycology is included for orientation. There are numerous excellent photographs of lesions, x-rays, and microscopic preparations. Each chapter concludes with a comprehensive bibliography.

This edition has been revised to include the rather large amount of information accumulated in the last 10 years, especially about the systemic fungus infections.—*H.R.*

Urology, Volumes I, II, III. By Meredith Campbell with 51 Contributing Authorities. Published by W. B. Saunders Company, Philadelphia. 2,356 pages, 1,148 figures. Price $60.

Many excellent textbooks of urology have been published since 1900 when urology became an accepted surgical specialty, and this new study edited by Dr. Campbell is one of the best. In 1926 Saunders published a similar set of volumes edited by the late Dr. Hugh Young which for many years was a standard textbook in urology. Twenty-eight years later this new text written by competent authors will again become a definitive reference text in urology.

The collaborating authors are all outstanding, and their contributions bring up-to-date the many developments of the last few years. An excellent integration with the basic sciences is achieved by the use of monographs by Doctors Pitts, Huggins, Moore, Ash, Engle, Hotchkiss, Jenkins, and others. Related fields are also covered by authorities. The field of urology has been greatly enriched by this splendid effort.—*W.L.V.*

Clinical Management of Behavior Disorders in Children. By Harry Bakwin and Ruth Morris Bakwin. Published by W. B. Saunders Company, Philadelphia. 495 pages, 14 figures. Price $10.

The authors of this book have succeeded in doing what has long been needed. They have gathered together in one book an extremely readable guide and reference book for the use of the general practitioner and pediatrician who must handle the great bulk of the behavior and emotional problems of children and often must be the source of referral for more specialized services. This book is also a valuable source of reference for the child psychiatrist and members of ancillary disciplines because of the ready availability of certain statistical information.

The book is rich in references to and abstracts from recent significant literature, and it is often from these abstracts that the above mentioned statistical material is taken. For example, in discussing cerebral palsy, McIntire's article on the subject is referred to, and his list of the frequency of the various etiological factors in cerebral palsy is cited. Another example of the useful information to which one might have need to refer frequently is the table setting forth the percentage of epileptics engaged in various occupations. Still another example is found in the chapter on sex education in which one finds a book list on sex education, books for children and adolescents or their parents.

Among the general attributes of the book should be mentioned the 34 pages of index which make for easy reference to specific topics.

The authors have included as their first section an excellent discussion of growth and development in which emphasis is placed on the emotional development of the child. The motor and mental capabilities at a given age are also adequately discussed.

The second section of the book takes up a discussion of general psychologic care from the newborn period through adolescence. The authors' point of view in regard to most matters of training, including early feeding techniques, is one of down to earth practicality with a notable absence of any rigidity.

The third section on the care of the physically ill and handicapped child takes up such matters as hospital care of children. The authors advise home care

UNIVERSITY OF KANSAS SCHOOL OF MEDICINE

Postgraduate Medical Courses

CLINICAL NEUROLOGY

January 24, 25, 26 & 27, 1955

FACULTY:

ADOLPH L. SAHS, M.D., Professor of Neurology and Chairman of Department, State University of Iowa College of Medicine, Iowa City.

D. BERNARD FOSTER, M.D., Lecturer in Medicine (Neurology); Director, Division of Neurology and Neurosurgery, Menninger Foundation, Topeka.

CHARLES E. BRACKETT, JR., M.D., Assistant Professor of Surgery (Neurosurgery).

J. de la FUENTE, M.D., Resident in Neurology.

FRANK A. MANTZ, JR., M.D., Assistant Professor of Pathology.

A. THEODORE STEEGMANN, M.D., Professor of Medicine and Chief, Section of Neurology.

WAYLAND A. STEPHENSON, M.D., Assistant Professor of Medicine (Neurology); Chief, Section of Neurology, Kansas City Veterans Administration Hospital.

GALEN M. TICE, M.D., Professor of Radiology and Chairman of Department.

ELLSWORTH H. TROWBRIDGE, JR., M.D., Assistant Clinical Professor of Medicine (Neurology).

A. B. WILLIAMSON, JR., M.D., Resident in Neurology, Kansas City Veterans Administration Hospital.

WILLIAM P. WILLIAMSON, M.D., Associate Professor of Surgery and Chief, Section of Neurosurgery.

RADIOLOGY AND RADIOACTIVE ISOTOPES

February 14, 15, 16 & 17, 1955

Guest Instructors:

LAUREN V. ACKERMAN, M.D., Professor of Surgical Pathology and Pathology, Washington University School of Medicine.

WILLIAM H. BEIERWALTES, M.D., Associate Professor of Internal Medicine, University of Michigan School of Medicine.

CARL C. BIRKELO, M.D., Clinical Assistant Professor of Radiology, Wayne University School of Medicine.

JAMES W. J. CARPENDER, M.D., Associate Professor of Radiology, University of Chicago School of Medicine.

CARL L. GILLIES, M.D., Professor of Radiology, State University of Iowa College of Medicine.

PHILIP J. HODES, M.D., Professor of Radiology, University of Pennsylvania Graduate School of Medicine.

JOHN F. HOLT, M.D., Professor of Roentgenology, University of Michigan School of Medicine.

B. V. A. LOW-BEER, M.D., Professor of Radiology, University of California School of Medicine.

JAMES STOBER, Physicist, Kelley-Koett X-Ray Corporation, Covington.

LEWIS G. ALLEN, M.D., Radiologist, Providence Hospital, K.C., Kan.

HILLIARD COHEN, M.D., Pathologist, Menorah Hospital, K.C., Mo.

DAVID S. DANN, M.D., Radiologist, Menorah Hospital, K.C., Mo.

HOMER L. HIEBERT, M.D., Radiologist, Topeka.

WM. M. KITCHEN, M.D., Radiologist, St. Mary's Hospital, K.C., Mo.

IRA H. LOCKWOOD, M.D., Radiologist, Research Hospital, K.C., Mo.

ANTHONY F. ROSSITTO, M.D., Radiologist, The Wichita Hospital.

SIDNEY RUBIN, M.D., Radiologist, Menorah Hospital, K.C., Mo.

ARTHUR B. SMITH, M.D., Radiologist, Research Hospital, K.C., Mo.

JAMES R. STARK, M.D., Radiologist, The Wichita Clinic.

JOHN W. WALKER, M.D., Radiologist, Research Hospital, K.C., Mo.



Woodcroft Hospital--Pueblo, Colorado

A private hospital for the scientific treatment of neuro-psychiatric disorders, including alcoholism and drug addiction. Beautiful landscaping and home-like surroundings afford a restful atmosphere. Accommodations vary from single rooms with or without bath to rooms en suite, allowing for segregation of guests.

Detailed information furnished on request.

KARL J. WAGGENER, M.D.

wherever possible. The care of the chronically ill child at home, and the care of such organic conditions as poliomyelitis, epilepsy, diabetes, and blindness are dealt with from the point of view of adequately handling the emotional problems involved.

Two sections deal with behavior disorders in children with consideration of the etiological factors and the diagnosis and treatment of these problems. There is a comprehensive presentation of the techniques of diagnostic evaluation using both interview and the projective techniques of psychological testing. The interesting and valid thesis is presented that children can tell a great deal verbally about their problems if properly approached. Psychotherapeutic techniques, including play therapy and adjunctive resources of foster home and institutional care, are briefly presented.

Two sections on the problems related to mental functioning and developmental abnormalities should give invaluable aid to the practitioner confronted with these problems in his practice, and they should serve also as a source of information and ideas should the practitioner be called upon to give talks to lay groups.

The sections on problems related to emotional development include chapters on abnormal emotional states, sex problems, and emotional deprivation in infants. Sections on problems of habit and training, organic disturbances with a large psychic component such as asthma, skin diseases and ulcerative colitis, anti-social behavior, and more specific syndromes such as schizophrenia in children are to be found in this book.

In general there is little to criticize, and the basic theoretical concepts and procedures are in line with current psychiatric thinking.

Last, but certainly not the least of the good features of this volume, is the list of references which follows each chapter.—*R.A.M.*

Government Spending

The fact that a man who earns $4,500 a year spends more than 2½ hours each day earning enough to pay his taxes, federal, state, and local, was one of the surprising statistics developed by the California Taxpayers Association in a recent survey. The private research organization made an analysis of the 90 billion dollars being collected by governmental agencies this year.

The same man, the survey reports, works only 1½ hours to pay for food for his family, about 1½ hours for housing, 36 minutes for clothing, and 42 minutes for transportation. Medical care is purchased with work completed in 23 minutes, recreation 20 minutes, and miscellaneous goods and services 23 minutes.



Nationally advertised Surgical Supplies and Equipment for your convenience at Topeka, Joplin, Kansas City, St. Joseph

GOETZE NIEMER CO

Traditions established during 60 years management by Dr. W. F. Goetze (AMA) assures intelligent servicing of your orders

THE SOUTHARD SCHOOL

Intensive individual psychotherapy in a residential school, for children of elementary school age with emotional and behavior problems.

THE MENNINGER CHILDREN'S CLINIC

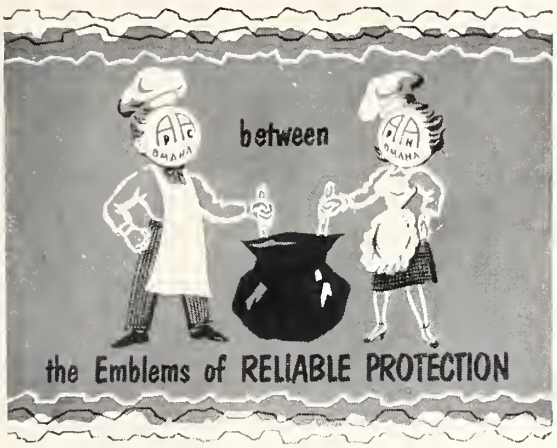
Outpatient psychiatric and neurologic evaluation and consultation for infants and children to eighteen years.

Department of Child Psychiatry

THE MENNINGER FOUNDATION

J. Cotter Hirschberg, M.D., Director — Topeka, Kansas; Telephone 3-6494

Something NEW is Cooking



MORE INSURANCE NOW AVAILABLE

think! HOW THESE AMOUNTS WOULD HELP IN PAYING ESTATE TAXES IN CASE YOU ARE ACCIDENTALLY KILLED . . .

$25,000.00 $20,000.00 $15,000.00 $10,000.00 $5,000.00

ALSO included in these plans . . . . EACH month for TOTAL DISABILITY either from ACCIDENT or SICKNESS

$500.00 $400.00 $300.00 $200.00 $100.00

SPECIFIC BENEFITS ALSO FOR LOSS OF SIGHT, LIMB OR LIMBS FROM ACCIDENTAL INJURY

HOSPITAL INSURANCE also for our members and their families

$4,000,000 Assets
$20,000,000 Claims Paid
52 Years Old

Physicians Casualty & Health Ass'ns.
Omaha 2, Nebraska

Medical and Surgical Supplies for Doctors of Medicine and Hospitals

Munns Medical Supply Co.
512 Kansas Avenue
Topeka, Kansas

Deformity Appliances of Quality

Orthopedic and Surgical Appliances

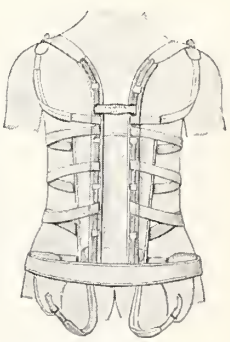
Artificial Limbs
Trusses

Abdominal Supports

Elastic Hosiery

Foot Supports

Surgical Corsets



Taylor Back Brace
Made to Order in
Our Own Factory

P. W. HANICKE MFG. CO.
1009 McGee St. Victor 4750
KANSAS CITY, MO.

Obstetricians Meet

A meeting of the Kansas State Obstetrical Society was held at the Muehlebach Hotel, Kansas City, on October 9, at the time of the refresher course in obstetrics at the University of Kansas Medical Center. Wives of members were guests. Dr. J. Curtis Lund of Rochester University was speaker of the evening.

Manual on Nursing Activities

A new manual designed to aid hospitals in making better use of personnel, *How to Study Nursing Activities in a Patient Unit,* was recently published by the Public Health Service, Department of Health, Education, and Welfare. The publication offers a method by which hospitals of all sizes may determine how nursing personnel time is distributed between duties requiring nursing skills and those which could be performed by other hospital personnel.

The purpose of the study is to give nurses more time to be with patients. Nursing personnel themselves have an opportunity to take part in the study and to analyze their own activities.

The foreword of the manual, written by Dr. Edwin L. Crosby of the American Hospital Association, states, "If many hours of nurses' time are being directed from their true purpose and spent in work others can do, this trend must be corrected. The problem is how to conserve professional nursing skills for their highest use. . . . Any hospital, large or small, can use the manual, adapting it readily to individual needs."

Copies of the booklet may be purchased from the Superintendent of Documents, Government Printing Office, Washington 25, D. C., at 25 cents each.

Rural Health Meeting

The 10th National Rural Health Conference sponsored by the American Medical Association will be held at the Schroeder Hotel, Milwaukee, February 24-26. Discussions will be held on the work of medical society rural health committees, responsibilities of citizenship, and training for rural practice.

CLASSIFIED ADVERTISEMENTS

FOR SALE—Modern well equipped air conditioned eye, ear, nose, and throat clinic located in red grapefruit section Rio Grande valley, Texas. Location ideal. Will sell equipment separate if wanted. Cheap. For details write Journal 22-54.

FOR SALE—Castle autoclave size 8 inches by 16 inches, practically new, priced right. Write the Journal 19-54.

WILL GIVE most all office equipment and furnishing to a younger doctor who will locate in my town of 3,500 population for general practice. Retiring because of age. Write the Journal 20-54.

LABORATORY TECHNICIAN, registered, wanted for midwestern medical group of eight. Excellent facilities and liberal salary. Write the Journal 21-54.



The Journal of the

KANSAS MEDICAL SOCIETY

INDEX TO VOLUME LV

JANUARY, 1954, TO DECEMBER, 1954, INCLUSIVE

Published Monthly by

THE KANSAS MEDICAL SOCIETY

INDEX TO VOLUME LV

In the list below, the number first mentioned identifies the month of issue.

Original Articles

Addison's Disease Complicated by Pregnancy: A Case with Postpartum Hypertension—Thomas J. Luellen, M.D., and Ray A. West, M.D., Wichita, Kansas 2-73

Alcoholics Anonymous—Mr. X 8-451

Arrhenoblastoma—G. E. Kassebaum, M.D., El Dorado, Kansas 12-703

Art of Medicine and the Cancer Patient, The—Ralph E. Jordan, M.D., Holton, Kansas 5-252

Carcinoma of the Cervix—L. A. Calkins, M.D., Kansas City, Kansas 7-377

Case of Actinomycosis of Ovary, A—Lyman C. Murphy, M.D., Wichita, Kansas 5-254

Complete Heart Block with Morgagni-Adams-Stokes Syncope Treated with Large Doses of Epinephrine—William E. Larsen, M.D., Kansas City, Kansas 5-249

Complications following Mitral Commissurotomy with Special Reference to Auricular Fibrillation—E. Grey Dimond, M.D., and Everton Marques dos Santos, M.D., Kansas City, Kansas 3-120

Congenital Absence of the Vagina: A Review and Case Report—Stanley L. VanderVelde, M.D., and Joseph J. Hovorka, M.D., Emporia, Kansas 11-628

Current Trends in Treatment of Congenital Megacolon—William N. Harsha, M.D., Oklahoma City, Oklahoma 10-569

Diagnosis of Lead Poisoning, The—John L. Morgan, M.D., Emporia, Kansas 1-11

Doctor Goes to Court, The—Joseph Cohen, LL.B., Kansas City, Kansas 7-388

Effect of Altitude on the Pathogenesis of Tuberculosis, The—William F. Casteen, Kansas City, Kansas 1-16

Error in Blood Sugar Determinations, An—Mark Dodge, M.D., Marion Mathy, Arthur W. Robinson, M.D., and Arnold V. Arms, M.D., Kansas City, Missouri 9-502

Experimental Production of the "Dry Field" in the Right Auricle and Ventricle—C. S. Stotts, M.D., and Stanley R. Friesen, M.D., Kansas City, Kansas..... 3-123

Fractures of the Anterior Tibial Spine—L. O. Litton, M.D., Kansas City, Kansas 3-117

Gallstone Ileus—Edward A. Scollin, M.D., Montgomery, Alabama 12-692

General Practitioner Studies Psychiatry, A—Prescott W. Thompson, M.D., Topeka, Kansas 11-624

Guillain-Barre Syndrome: Report of Two Cases Treated with Vitamin B Complex, Cortisone, and ACTH for Relief of Pain—Thomas L. Foster, M.D., and Charles Pokorny, M.D., Halstead, Kansas 5-256

Hypersplenism Associated with Leukemia—Lee Hanes, M.D., Kansas City, Missouri 12-697

Hypoprothrombinemia-Inducing Activity of Warfarin Sodium (Coumadin Sodium), The—Shepard Shapiro, M.D., New York, New York 12-687

Immunity and Resistance in Poliomyelitis: A Brief Résumé of the Literature—Herbert A. Wenner, M.D., Kansas City, Kansas 9-495

Low Salt Syndrome in Children during Hot Weather, The—Mary E. Evans, M.D., Kansas City, Kansas ... 3-125

Lung Cysts—Melvin A. Rabe, M.D., Leavenworth, Kansas 9-489

Management of Acute Laryngotracheobronchitis—Harold L. Hickey, M.D., Denver, Colorado 1-7

Management of Traumatic Rupture of the Left Superior Pulmonary Vein, The—Harry R. Custer, M.D., Colby, Kansas, and Karl P. Klassen, M.D., Columbus, Ohio 10-563

Massive Diuresis in Congestive Heart Failure—Samuel Zelman, M.D., Topeka, Kansas 6-317

Medical Care in Pediatric Surgery—Frederic G. Burke, M.D., Washington, D.C. 11-621

Modern Concept of Otitis Media—Harold L. Hickey, M.D., Denver, Colorado 2-63

Multiple Primary Malignant Lesions—Jack W. Welch, M.D., Halstead, Kansas 6-314

Paroxysmal Ventricular Tachycardia Occurring during a Two-Step Exercise Test: Report of Case—Robert A. Jordan, M.D., Kansas City, Kansas 10-566

Pathology of Ectopic Pregnancy, The—Fred A. Brandt, Kansas City, Kansas 2-66

Poisonings with the Newer Pesticide Compounds—Thomas R. Hood, M.D., and Evan Wright, Topeka, Kansas 7-384

Report on the Tubeless Gastric Analysis—R. L. Van Citters, M.D., and Mahlon Delp, M.D., Kansas City, Kansas 6-311

Rupture of the Colon Secondary to Muscular Strain: Report of Two Cases—W. G. Cauble, M.D., Wichita, Kansas 9-500

Sodium Pentothal Solution, An Adjuvant in the Treatment of Acute Alcoholism—DeMerle E. Eckart, M.D., Hutchinson, Kansas 8-453

Spontaneous Intravascular Clotting—William W. Abrams, M.D., Kansas City, Kansas 7-379

Treatment and Management of Alcoholics in a General Hospital—Thomas L. Foster, M.D., Halstead, Kansas 8-441

Treatment of Diabetic Acidosis and Diabetic Coma, The—Edward Tolstoi, M.D., New York, New York 1-1

Treatment of the Acute Alcoholic from a General Practitioner's Viewpoint—Z. Miles Nason, M.D., Kansas City, Kansas 8-436

Treatment of the Alcoholic Patient in Kansas State Mental Institutions—George W. Jackson, M.D., Topeka, Kansas; Earl F. Morris, M.D., Larned, Kansas; J. E. C. Morton, M.D., Osawatomie, Kansas, and Stuart-Averill, M.D., Topeka, Kansas 8-446

Twenty-Four Year Review of Cesarean Sections at Kansas University Hospitals, A—J. C. Schroll, M.D., and L. A. Calkins, M.D., Kansas City, Kansas 3-131

Use of Hydrocortisone-Neomycin Ointment in the Treatment of Various Dermatoses, The—Donald L. Fuhrman, M.D., and Bruce V. Drowns, M.D., Kansas City, Missouri 6-322

Use of Trypsin in Rheumatoid Arthritis, The: A Clinical Study—William H. Goodson, Jr., M.D., Donald L. Rose, M.D., and William S. Alyea, M.D., Kansas City, Kansas 3-129

Value of Gastroscopy, The—Henry Laurens, Jr., M.D., Salina, Kansas 10-559

Weber-Christian Disease: A Case Report—Charles Pokorny, M.D., and C. Alexander Hellwig, M.D., Halstead, Kansas 2-70

Widening Scope of the State Hospital with Special Reference to Geriatric Medicine and Surgery, The—C. Maxwell-Joyner, M.D., Larned, Kansas, and Rudolf Hofbauer, M.D., Wichita, Kansas 11-631

INDEX TO VOLUME LV

In the list below, the number first mentioned identifies the month of issue.

Tumor Conferences from the University of Kansas Medical Center

Adenocarcinoma of the Prostate 1-25

Biopsy Procedures in Office Practice 12-712

Hemangiomas and Lymphangiomas 6-338

Management of Incurable Cancer, The 8-460

Parotid Tumors 3-144

Recent Trends in Therapy of Carcinoma of the Breast 10-580

Senior Theses from the University of Kansas School of Medicine

Adverse Effects of Penicillin Therapy—W. D. Bradshaw, M.D. 9-536

Anticoagulants, The: Heparin and Dicumarol—Clarence E. Thompson, M.D. 6-352

Cerebral Circulation, The: Comments on Cerebral Angiospasm—John O. Mansfield, M.D. 2-92

Complications of Polycythemia Vera, The—Curtis A. Nystrom, M.D. 7-399

Medical Management of Peripheral Arteriosclerosis—John D. King, M.D. 10-592

Parkinson's Disease—Glen G. Halliday, M.D. 12-721

Pathogenesis of Hypochromic Microcytic Anemia, The—David L. Zacharias, M.D. 3-164

Physiological Theories of the Etiology of Alcoholism—Robert D. Parman, M.D. 8-474

Radioactive Iodine in Hyperthyroidism—Lowell Rhodes, M.D. 1-40

Some Basic Concepts of the Acute Radiation Syndrome—Eugene T. van der Smissen, M.D. 11-656

Treatment of Hepatic Insufficiency—Helen Jones, M.D. 5-278

Editorials

Age and Distribution of Doctors 12-711

A.M.A. Annual Session 9-508

Annual Meeting in Hutchinson 11-636

Approach to Problems of Alcoholism 8-457

Change in A.M.A. Dues Policy 1-23

Chiropractors Withdraw Petitions 6-325

Doctors at War and at Home 2-80

Eighth Annual K.U. Issue 3-139

Events in Washington 2-80

Facts of Life, The 1-23

Federal Aid in Kansas 10-577; 11-635; 12-707

Federal Health Programs ..... 7-395, 8-457 and 9-507

History of the Society, A 9-507

Honor in the Practice of Medicine 5-262

Is It Art? 11-635

Kansas Medical Day 3-140

Malpractice Insurance 7-396

Maternal Mortality Study 10-577

Memorial Fund 2-81

Message From the Auxiliary, A 12-710

New Council, The 6-326

New Year's Resolutions 12-707

Orgone Energy 5-262

Physician Placement 7-398

Polio Vaccination Field Trials 1-21

Prehistoric Medicine 2-79

Progress at the Medical School 3-139

Referrals to the Medical Center 6-327

Thanks, Jackson County 6-328

Vaccine Evaluation Phase 8-458

VA Non-Service Connected Cases 1-22

Veterans' Affairs 5-261

United Funds 10-578

Vaccinia Immune Gamma Globulin 2-81

Death Notices

Bence, Dr. Arthur Edgar 12-720

Boudreau, Dr. Clarence 7-424

Fowler, Dr. Joseph 12-720

Haerle, Dr. Edward Jacob 2-84

Kilbourn, Dr. Edward D. 6-372

Kirby, Dr. George Wightman 12-720

Meisburger, Dr. Richard G. 7-424

Menninger, Dr. Charles Frederick 1-32

Morris, Dr. Benjamin Smith 12-720

Nicoll, Dr. David Thompson 10-591

Robb, Dr. James Carey 11-649

Settle, Dr. James Albert 3-153

Sharpe, Dr. Orlan Dayton 6-372

Siever, Dr. Charles Moses 2-84

Thacher, Dr. Mowry Stafford 6-372

Walker, Dr. Oliver David 11-649

Regular Headings and Sections

Activities of Members 1-33; 2-82; 3-152; 5-272; 6-346; 7-404; 8-469; 9-528; 10-590; 11-650; 12-719

Announcements 1-38; 2-108; 3-172; 4-242; 5-306; 6-343; 7-424; 9-530; 10-608; 11-674; 12-720

Blue Shield 1-39; 2-77; 3-142; 5-274; 6-350; 7-406; 8-472; 9-532; 10-589; 12-711

Book Reviews 1-54; 3-178; 5-298; 7-422; 9-509; 10-610; 11-680; 12-730

Clinicopathological Conferences 2-85; 5-264; 7-408; 9-519; 11-638

County Societies 1-35; 2-83; 3-149; 5-274; 6-342; 7-420; 9-532; 10-591; 11-652; 12-718

Kansas Press Looks at Medicine, The 1-50; 2-84; 3-174; 6-351; 7-420; 11-654

Month in Washington, The 1-37; 3-137; 5-276; 6-368; 7-393; 8-455; 10-575; 11-637; 12-705

President's Page 1-20; 2-78; 3-138; 4-206; 5-260; 6-324; 7-394; 8-456; 9-506; 10-576; 11-634; 12-706

Service Separations 5-263; 6-327; 7-397; 8-459; 10-579

44277
Kansas medical society. Journal
AUTHOR
v.55, 1954
TITLE
DATE DUE

Kansas medical society. Journal
v.55, 1954
4
RETURN THIS BOOK ON OR BEFORE LAST DATE STA